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TEXAS State Journal of Medicine

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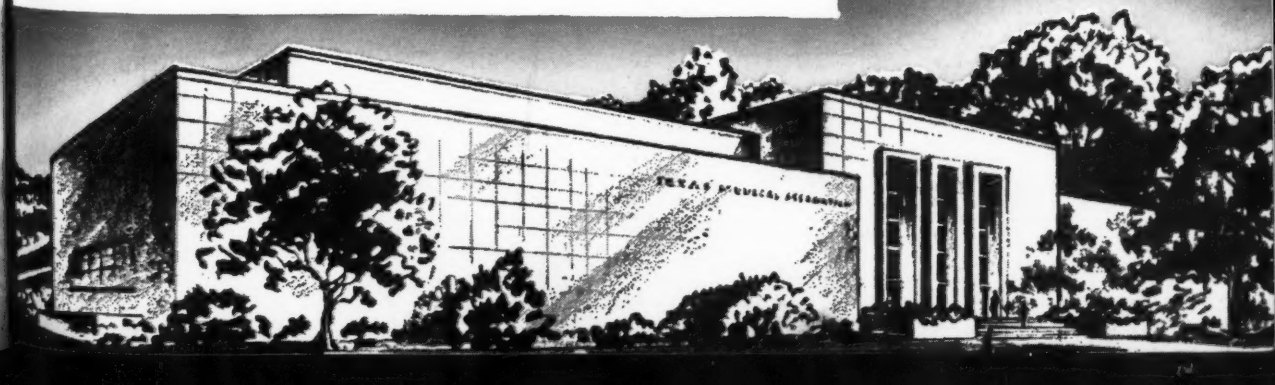

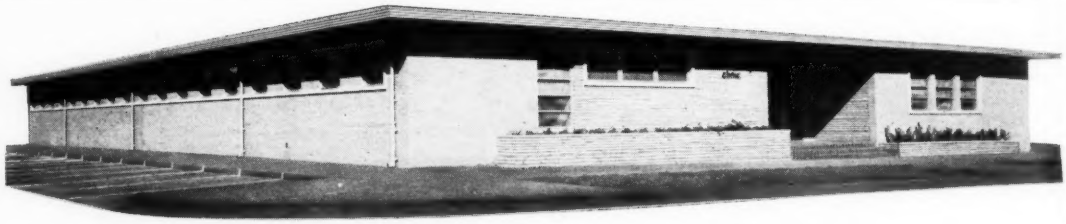

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President's Page

WOMAN'S AUXILIARY

The Woman's Auxiliary to the Texas Medical Association held its first meeting May 13, 1918, at San Antonio, and here the first auxiliary to a state medical society was organized. These pioneering doctors' wives sold the "Auxiliary" idea to women in other parts of the country. On motion of the Texas Delegation, the Woman's Auxiliary to the American Medical Association was organized in 1922. Perhaps this Texas heritage helps explain the spirit and vitality of our sister organizations. In any event, the record of the Auxiliary's first thirty-five years is one of steady growth and accomplishment, sometimes in the face of amused tolerance or downright opposition from physicians.

The supreme devotion of our Auxiliary members to the cause of organized medicine and their willingness to serve are reflected in the manner with which their activities are carried out. These women are serious about their Auxiliary work, and on the basis of their record, they deserve to be treated seriously. Sixty-three thousand women working to better the health of our people and to enhance the reputation of the medical profession is a force worthy of respect.

One of the original aims and still an important aim of the Auxiliary is to encourage friendship and good will among the families of physicians. In a profession so dependent upon mutual cooperation and the free interchange of scientific knowledge, friendship is more than a personal asset. In recent years political attacks upon physicians have made unity more necessary than ever. The promotion of friendship among the families of physicians is, therefore, an increasingly valuable contribution of the Auxiliary.

Aid to medical education through contributing to the American Medical Education Foundation is an important interest to almost all Auxiliaries. A total of $51,000 has been given to this fund during the past three years. Over $30,000 was contributed last year.

Major emphasis by all Auxiliaries has been placed upon nurse recruitment. To arouse the interest of high school girls in such a career, Auxiliaries have entertained at nurse recruitment teas, organized tours of local hospitals, sponsored showings of films such as "Girls in

White" and distributed quantities of literature on nursing. Future Nurses' Clubs are sponsored by many Auxiliaries. Once interest has been aroused, finances sometimes prove a problem to a would-be nurse. Again the Auxiliary is ready to lend a helping hand. Last year twenty-four states granted loans amounting to $29,000 to 212 girls, and twenty-seven states awarded scholarships totalling $53,000 to 351 girls. Thus in just one year, the Auxiliaries assisted 563 girls to enter nurses' training!

Moving beyond these intraprofessional projects, the Auxiliary has turned to the health needs in American communities. Members work actively in hospital campaigns and in annual fund drives for heart disease, infantile paralysis, arthritis and rheumatism, tuberculosis, cancer, and others. Their influence for good health is again felt through P-TA's and school boards. Adult health education also claims Auxiliary attention. Members actively promote the sale of subscriptions to *Today's Health* and sponsor community "Health Days."

Auxiliaries have sponsored the showing of civil defense films before school children and community groups. Members have enrolled in first aid and home nursing courses to prepare themselves for useful roles in an emergency. Some qualified members have taught such courses. Others are serving as sky watchers and assisting with blood donation programs. Some members are found serving on local defense boards.

The Auxiliary participates in other activities upon invitation for guest representation at the annual meetings of such organizations of note as the American Cancer Society, National Advisory Committee on Health Councils, National Foundation for Infantile Paralysis, Assembly of Woman's Organizations for National Security, Advisory Committee on Woman's Activities, National Foundation for Mental Health, and President's Safety Council.

This is a condensed report of what our wives are helping to accomplish. I think we doctors owe them a vote of gratitude. Few other men can claim such sincere and devoted co-workers! Buy *Her* a Christmas present. MERRY CHRISTMAS.

George Turney M.D.

PTR diagnotes

REPORT OF PUBLIC RELATIONS COMMITTEE: January 23 County Society Presidents and Secretaries meeting program supplemented to include panel on Veterans' Medical Care - includes three thirty-minute talks by competent physicians followed by one hour of questions and answers. Session will have advantage of wide legislative background of Dr. Frank Wilson, Washington AMA office director.

Presidents and secretaries present will be given complete AMA information kit on VA medical care, content of which offers basis for AMA policy on hospitalization entitlement of veterans. AMA information kit includes factbooks on VA medical care, speeches, radio interviews, AMA policy statements, reprints of editorial opinions, and the address "Veterans' Medical Care - Dole or Dividend?" delivered by AMA President Edward J. McCormick before the Rocky Mountain Radiological Society.

It is hoped that meeting will provide officers with complete information regarding the facts on veterans' medical care. Should be best yet. Four mailing pieces will keep those interested reminded to attend.

On another important project: Not one request for doctors from communities in District 7 following test survey on doctor shortage. Offers for assistance from Texas Medical Association went out two months ago to all towns 500 and above population.

Requests sent out by Committee on Public Relations to all county societies for information on various projects were well received. Committee urges continuation of efforts to keep central office informed.

Use of Association's Memorial Library continues to improve as more doctors become informed of real value of asking it for materials for papers, speeches, programs. Personalized service one of the best.

ON THE NATIONAL SCENE: AMA Washington Letter reports that present Defense Department planning envisions no extension of the doctor draft beyond July 1, 1955, but instead it calls for a program of "fence mending" and "belt tightening," as stated by federal officials concerned with the law at the annual meeting of the Association of Military Surgeons. This program was outlined at the same time that Dr. Edward J. McCormick, AMA President, told the surgeons: "It is our belief that this is a most propitious time for devising a program which will clearly eliminate any need for this legislation well in advance of July 1, 1955."

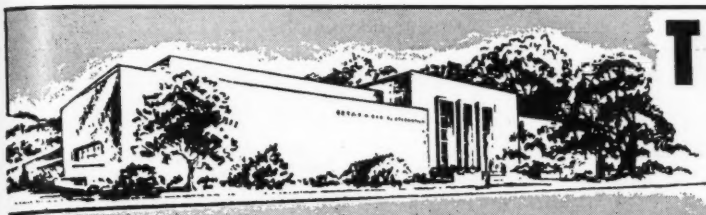
The Veterans Administration, by regulation, has changed the form that veterans are required to fill out when they apply for hospitalization for nonservice connected conditions. Previously, a veteran applying for hospitalization was required to answer only "yes" or "no" to the question, "Are you financially able to pay the necessary expense of hospitalization or domiciliary care?" If the answer was "no," he was eligible and was asked no more questions. In the future, if a veteran answers "no" to this question, he will then be required to answer additional questions concerning his financial status.

AMA Washington Office has sent out special report No. 13, which explains this VA development completely with copies of the new form. This information should be valuable in that the Washington report not only includes changes VA is making, but also explanation VA is giving for the changes. Copies can be secured from central headquarters in Austin.

FROM OVER THE STATE: Harrison County Society doing its part to keep doctors of East Texas and West Louisiana abreast of medical advances. Sponsoring a series of monthly talks by experts imported from larger medical centers. One address suitable for wives also included to round out program.

Bexar County Society reports one of its members brings a morning paper to each of his hospitalized patients on his early rounds. Very good PR.

Harris and Galveston County Societies-Texas Medical Association jointly sponsored Medical Students Days reported highly successful December 11 and 12.



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Volume 49 **Austin, Texas, December, 1953** **Number 12**

PUBLICITY OR PUBLIC INTEREST?

The names of Rockefeller and Rosenwald, Carnegie and Kellogg have become almost household words because of the beneficent foundations which they established years ago. Perhaps it is with the names of these men and their trusts for the public welfare in mind that certain persons today organize "foundations" for a variety of medical and scientific purposes —each foundation bearing the name of its instigator. With those who have a real sense of public service and direction and who recognize the value of working through already established institutions and agencies to achieve worth-while goals with little reference to personal credit there can be no quarrel. We can be thankful that men and women blessed with a large share of this world's goods see fit to dedicate a portion of it for the benefit of their fellows; notable examples of such philanthropies exist in our own Southwest, and we can be proud of them.

Some, however, apparently sieze on the first likely splinter on which to burn their name for posterity, inviting strangers from far and near to make donations and ignoring the more sturdily and comprehensively planned structures to which the splinter might otherwise offer substance. The broad, well-recognized agencies may lose unified support at the expense of small, ill-defined, poorly-directioned foundations apparently designed primarily for personal publicity, sometimes secondarily to lessen a tax burden. That is not to say that all motives behind such foundations are selfish or bad; undoubtedly a modicum of concern for humanity helps to throw the balance toward organization of most scientific or medical foundations, but this concern may be unenlightened.

The same desire for publicity may motivate a clinician or researcher to push a new drug, an individualized surgical technique, or a personal invention beyond the point of usefulness, and if a word of restraint is offered, pride or stubbornness may dictate a still stronger stand in favor of the pet discovery.

Physicians need to be cautious in accepting at face value pleas for recognition or support

of previously unheard of foundations, especially coming from distant places and therefore difficult of investigation and evaluation, just as traditionally they have been reluctant to accept new therapeutic agents until their worth has been proved. Unquestionably some of these foundations have been established in the best of faith and perhaps are doing no harm in themselves. At the same time, as they scatter the interest and support of the public, emphasizing one aspect of a basic problem to the exclusion of other equally important phases, all of which may come within the purview of a nationally recognized agency, they may deter progress toward solution of some of the major health problems of the day in our country.

A Prayer for Christmas

Lord, make me an instrument of your peace.
Where there is hatred, let me sow love.
Where there is injury, pardon.
Where there is doubt, faith.
Where there is despair, hope.
Where there is darkness, light.
Where there is sadness, joy.

St. Francis

PRINCIPLES INTO PRACTICE

"Only . . . physicians . . . who subscribe to the Principles of Ethics of the American Medical Association shall be admitted to membership," according to the Constitution of the Texas Medical Association. Yet how many members of the organization know exactly what this entails?

For those who may never have read the Principles of Medical Ethics in their entirety or who may wish a copy for ready reference, this guide to professional action is reprinted in the Organization Section of this JOURNAL. As the preamble states, in most instances "the physician who is capable, honest, decent, courteous, vigilant, and an observer of the Golden Rule, and who conducts his affairs in the light of his own conscientious interpretation of these principles, will find no difficulty in the discharge of his professional obligations." In this day of criticism of the medical profession, each doctor should be certain in his own mind that he lives up to the aforementioned qualifications.

"AS BIG AS THE MAP OF TEXAS"

Tuberculosis still casts a shadow as big as the map of Texas, say tuberculosis association authorities. But that shadow is being lightened through extended educational programs for physicians and public, increased support of research, improved methods af case finding and treatment, and better public health measures for control of the disease.

One of the greatest needs as far as the medical profession is concerned is for more adequate reporting of tuberculosis. It is of prime concern to all who deal with the disease—the general practitioner and his specialist colleague, the public health official, the welfare worker, the tuberculosis association board member. Although the death rate from tuberculosis is declining rapidly, morbidity statistics leave no doubt but that the disease still claims victims at a steady pace. For example, new cases reported for Texas in the years 1950 through 1952 were as follows: 4,618; 4,415; and 4,385, in that order. Through November 21 of this year 3,603 new cases have been recorded.

Physicians, as scientists, citizens, humanitarians, and philanthropists, will continue to wage war against this leading public health problem by contributing generously to the Christmas Seal sale now in progress.

CURRENT EDITORIAL COMMENT

A CURE FOR A SICK MEDICAL PROFESSION

I am not a practicing physician in the sense of having a sign over my office, a receptionist, and posted office hours. I am a college doctor, and as such I, figuratively speaking, am viewing the busy life of physicians in private practice from the sidelines. My work is mostly office work, but I am in contact almost daily with the patients of private practitioners, general or specialized. Some patients return to me after I have sent them to other doctors; others tell me stories, unsolicited, of their experiences in a busy doctor's office. Many are bewildered by technical information they receive about symptoms. Some of my informers are not patients but parents of students who have sent their children to the family physician or specialist on my recommendation or on their own initiative.

The patient receives medical attention, drugs, and explicit instructions on how to take his medicine, but he knows little or nothing about what disease he is suffering from; how long he must stay in bed, be out of college, or take his medicine; whether his disease is contagious in his home where there are other children; what the dangers of convalescence are; and many other questions which should be settled.

If it is a surgical case, many of which I refer to surgeons, the patient is seen, roentgen-rays are made, and the patient is told an operation is necessary as soon as possible. How long he is likely to be in the hospital, how soon he may expect to return to school, the estimated cost of the operation, the probable prognosis, and other problems of interest to one about to undergo surgery are not discussed with the patient.

As I am writing, the magazines, journals, and newspapers are ablaze with criticism of the evils of overspecialization, fee splitting, unnecessary medication and surgery, and the neglect by doctors to give patients the time and personal attention they pay for.

After thirty years as a college physician, catching the repercussions of public criticism of our profession in addition to my daily work of caring for 2,700 students, I am inclined to believe that much of the criticism is justified. Never in history has the art of medicine moved forward at so rapid a pace as at the present time. Men and women who have chosen medicine as a profession are straining every nerve to keep apace with medical progress. At the same time the high cost of living has caused a nervous tension and hysteria which have permeated every business enterprise. This complex has raised incomes but has inflated prices of every commodity far beyond the compensating incomes. This vicious circle of living has rolled into the doctor's office, and overcharging by the physician is but an effort to keep up with the procession. But the medical profession is dealing with human lives, not stocks and bonds, and with human beings broken in body and spirit.

This type of patient needs something besides cold diagnosis and stereotyped treatment. He needs sympathy and explanation of his difficulties in nontechnical terms. At times he even needs advice and counsel on matters not directly bearing on his disease or deformity. He needs a little more personal attention to give him courage and confidence. These things he is not getting in the busy, overcrowded doctor's office today. Too much is left to the receptionist—even the amount of the fee in some cases. There is too much of the "get 'em in, get 'em out" spirit in medical practice today. Some of the personal interest, at least apparent if not real, of the old-time doctor is badly needed.

The late Dr. William Osler, one of the greatest physicians of them all, said, "Know your patient first, and the disease afterward," and our own Hippocratic Oath stresses the curing of the patient, and not the fee, as of greatest importance in medical practice.

B. F. JENNESS, M. D., Director of Health Service, Texas Western College, El Paso, Texas.

ORIGINAL ARTICLES

MANAGEMENT OF CLEFT LIP DEFORMITIES

SANFORD GLANZ, M.D., Corpus Christi, Texas

THE treatment of cleft lip deformities has been greatly advanced in recent years by improvements in surgical technique and pediatric management. A step-line closure described by LeMesurier[12] has marked significant progress in the surgical repair. Results with this procedure have removed the stigma of the vertical straight-line harelip scar. Other advantages include improved nostril contour, bilateral symmetry, a normal cupid's bow, and eversion with fullness of the vermillion border. The technique also has been adapted to secondary revisions of unsightly harelip scars. Overall treatment of harelip deformities, however, still presents a challenge because of frequency of occurrence and the problems of care and surgical correction.

Incidence of this deformity, as reported from nationwide vital statistics, averages 1 out of every 750 live births. Statistical data available in Texas, covering the past five years, give an approximate ratio of 1 to 700 live births.* The recorded incidence includes clefts of the lip and palate alone or in combination. This compares similarly with other statistics given throughout the country, including the figures by Brown of 1 to 665 in Missouri[5]; Slaughter, 1 to 770 in Wisconsin[17]; and Ivy, 1 to 762 in Pennsylvania.[11] Frequency by sex has shown the greater number occurring among male infants. Oldfield[14] in great Britain gave a ratio of 3 males to 2 females. In Texas, the ratio of boys to girls is approximately the same.* There is also a greater frequency among the white population. Davis,[8] in Baltimore, reported an incidence in the Negro population one-half as great as that in the white race, 1 to 1,788. Unilateral cleft lips are much more common than bilateral clefts, and occurrence on the left side is noted to be twice as frequent as on the right. Cleft palate accompanying cleft lip is apparent in about 50 per cent of these patients.[19] Club foot is the only other congenital anomaly that has been proved more common than facial clefts.

Embryologically, the cleft is generally believed to develop at the time of fusion of the maxillary and frontonasal processes in about the sixth to eighth week of gestation. Failure of these processes to fuse will cause cleft deformities in varying degrees. Other theories of cleft formation have been advocated. Recently, Orban[15] proposed the theory that ectodermal fusion takes place but that there is a failure of mesenchymal growth across the gap. Accordingly, the cleft develops as the ectodermal union resolves. Clinical observation of thin webs of epithelium across the cleft and of partial clefts without maxillary involvement gives some support to this viewpoint.

Although the causes of cleft lip are not categorically known, two factors have been shown to play a significant role, that is, inheritance and maternal malnutrition or disease during the first trimester of pregnancy. In a thorough study of the hereditary influence, Fogh-Andersen[9] showed that 30 to 40 per cent of these patients had near relatives with similar deformities. The influence of maternal malnutrition has been established through extensive animal research by Warkany.[20] Other etiologic theories proposed concern the effects of increased maternal age, hormonal imbalance, and exposure to roentgen rays. It may be noteworthy that the Rh factor does not appear to play any role in the formation of this abnormality.

With current advances in physiologic studies, there is reason to believe that the causes may become more clearly understood. Until such time, we can only conjecture and advise the inquiring parent as to the hereditary possibility. If two apparently normal parents have a child with cleft lip, however, and there is no previous familial history of this deformity, they can be reasonably assured that chances of a similar abnormality occurring in a future sibling are less than 5 per cent.[14] The incidence, of course, tends to increase if one or both parents have the same malformation.

Various procedures have been devised throughout the years to improve the reparative technique for the infant cleft lip. The common aim has been to obtain adequate muscle union of the repaired cleft, normal width to the floor of the nose, and restoration of contour to the lip and vermillion border. The various methods and modifications of straight, curved, and broken-line closures have been discussed by Davis[7] in a collective review of unilateral cleft lip repairs. The straight and curved-line techniques include those described by Mirault (1844), Rose,[16] Blair,[3] and Brown and McDowell.[4] Of more pertinent relationship to

Read before the Section on Pediatrics, Texas Medical Association, Annual Session, Houston, April 29, 1953.

**Data obtained from the Bureau of Vital Statistics, Texas State Department of Health.*

this paper is the evolution of the broken-line closure, which is the procedure I favor. The use of the zigzag scarline was first described by Hagedorn in 1884, and again in 1892.[10] LeMesurier devised modifications of this procedure, which he described in 1949[12] and 1952.[13] The LeMesurier technique altered the zigzag scarline into a step-line closure, utilizing a square flap of tissue from the lateral side of the cleft. Bauer, Trusler, and Glanz[2] described further modifications in 1953. The results of this procedure have proved equally successful in both complete and incomplete clefts of the lip.

In the repair of bilateral cleft lips, various adaptations of the rectangular flap have been described by Brown[6] and Barsky.[1] These techniques generally enable a one-stage closure. However, two-stage procedures are occasionally necessary. It is also important not to disturb the maxillary segments, for interference with these growth centers may be detrimental to normal development of the upper jaw. Correction of the double cleft deformity is more difficult, and it is still apparent that results from any technique have not yet reached the standards of the unilateral repair.

PRELIMINARY CARE

The problem of revealing and explaining a harelip deformity to the family is one that should be handled with considerable foresight and tact. Unfortunately, the experience of laymen with this problem makes them ill-prepared to understand and accept the improved prognosis for infants with cleft lip. Many of these clefts in earlier generations were corrected improperly and left the facial stigma of the harelip cripple. "Granny" tales and old superstitious beliefs sometimes will lead a parent subconsciously to reject a child that is different. The pediatrician can counsel the parents so that their psychologic viewpoint is sound and well adjusted from the start. Explanations of modern surgical advances assuring a successful repair usually satisfies the family that the infant will grow into a normal and healthy child. Whenever possible, the physician who has given the prenatal care and has the confidence of the parents should join in counseling. It is sometimes beneficial to hospitalize the baby until time for surgery to avoid the effect on the parents of curious family, friends, and neighbors. However, this is an expensive means of psychologic adjustment and not often recommended.

In the immediate care of the infant, feeding and nutritional maintenance are the factors to be considered. Breast feeding is not advisable because of contemplated early surgery and the difficulties that do arise in the mechanics of feeding. Actually, these infants can suckle and, with care and patience on the part of the informed and persevering mother, the feeding problem can well be minimized. Many infants nurse well from a bottle with an enlarged puncture in the nipple. When this appears inadequate, however, an asepto-bulb syringe with a short length of rubber tubing in my experience has proved satisfactory. Other simple but effective measures include the standard medicine dropper and the ordinary teaspoon. Regardless of the type feeder used, the ultimate endeavor is to deliver the required milk directly to the baby's throat. The mechanism of swallowing is not impaired. Infants with cleft lip and palate, however, are subject to choking spasms. A semirecumbent position in the arm of the person feeding will prevent abnormal distribution of fluids and avoid untoward complications. Gavage feedings have been advocated, but the pharyngeal and esophageal trauma frequently involved in tube feeding the newborn infant may contraindicate this procedure.

A regularly prescribed formula usually will prove adequate. However, nutritional deficiencies may require alteration of dietary intake. Vitamins, iron, and increased nutrition will aid in preparing the baby for surgery. Supplemental polyvitamin preparations should include excessive doses of ascorbic acid. Vitamin K therapy, given as a separate entity, aids in correcting the low prothrombin levels frequently present at birth. Iron requirements are based on hematologic determinations. A hemoglobin level of less than 75 per cent should be a contraindication to surgery. This can be remedied by iron therapy and, in more severe deficiencies, by small blood transfusions. Antibiotic therapy is used prophylactically in recommended dosages.

PREOPERATIVE CONSIDERATIONS

The time for surgical repair should be agreed upon by the pediatrician and surgeon as soon as the infant is gaining weight and appears to be physiologically stable. A hurried, perfunctory closure immediately after birth will endanger the infant's life and may lead to a disappointing result. It should be emphasized that this anomaly does not constitute an emergency, and, as in all elective procedures, the operation should be postponed until the infant's overall condition warrants surgery.

The safe surgical correction of cleft lips within four to six weeks after birth has been made possible by advances in pediatric care, anesthesia, and operative technique. In unilateral clefts, surgery may be undertaken when the baby has reached a minimum weight of 7 pounds. Double clefts of the lip require a more formidable procedure, and the baby should be older and weigh at least 9 to 10 pounds.

Certain other factors should be considered in determining the time for surgery. The absence of fever

and respiratory or intestinal infection is, of course, mandatory. A careful physical examination is always routine in ruling out any concurrent congenital defects. An infant with major cerebrospinal, intrathoracic, or intra-abdominal congenital deformities associated with cleft lip or palate is a poor surgical risk. In such cases, the importance of surgical postponement and observation cannot be overemphasized. Other types of concurrent congenital deformities usually do not contraindicate early repair of the cleft lip.

Closure of the cleft within the first two months is important when the alveolar ridge is involved. Pressure of the closed lip against the malpositioned alveolus will help mold the maxilla before extensive calcification has occurred. This applies especially to a complete bilateral cleft lip with a protruding premaxilla and prolabium. Muscular compression of the repaired lip will tend to reposition the premaxilla into its normal relationship with the main maxillary arch.

SURGICAL PROCEDURE

Immediate preoperative preparation includes the usual withholding of formula and water. A three hour abstinence, however, is sufficient, for a longer period will produce untoward effects. These infants become dehydrated quickly and will have a rapid elevation in temperature if fluids are withheld too long.

General insufflation anesthesia is safe and satisfactory in almost all cases. Endotracheal intubation can be used in the hands of the thoroughly experienced. However, the danger of laryngeal edema necessitates more extensive postoperative surveillance in such cases. Although a unilateral harelip operation is not considered a formidable procedure, the blood loss in an infant may be of significance. For this reason, regional hemostasis is effected by the local injection of an appropriate amount of epinephrine in procaine solution.

The surgical procedure, according to the LeMesurier technique, involves the outline of a quadrilateral flap in the lateral part of the lip that will fit a reciprocal defect in the central portion of the lip. Adjacent soft tissues of the face are widely undermined to free all attachments to the bony structures so that closure without tension can be effected. The nasal cartilage is freed from the skin to allow rotation of the flattened alar rim into a tube. Mobilization of the columella will permit its repositioning to the midline. The cleft

FIG. 1. Unilateral cleft.
Left. Preoperative view showing a right cleft involving the lip and alveolus.
Right. Postoperative view showing the result twelve months later.

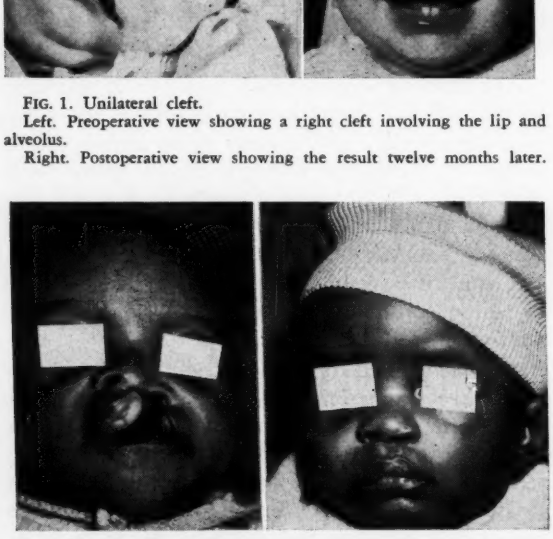

FIG. 2. Wide complete unilateral cleft.
Left. A wide left cleft involving the nose, lip, maxilla, and palate Note flattening of the alar rim.
Right. Postoperative result when the child was 10 months of age. Nostril symmetry has been restored.

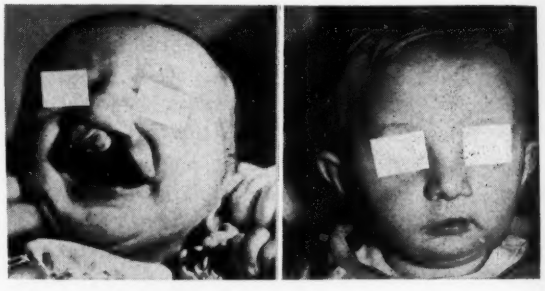

FIG. 3. Wide complete unilateral cleft.
Left. A 4 week old infant with extremely wide left cleft involving the nose, lip, maxilla, and palate. This was repaired when the child was 5 weeks of age.
Right. Eighteen months later, adequate development of lip and nostril contour is evident.

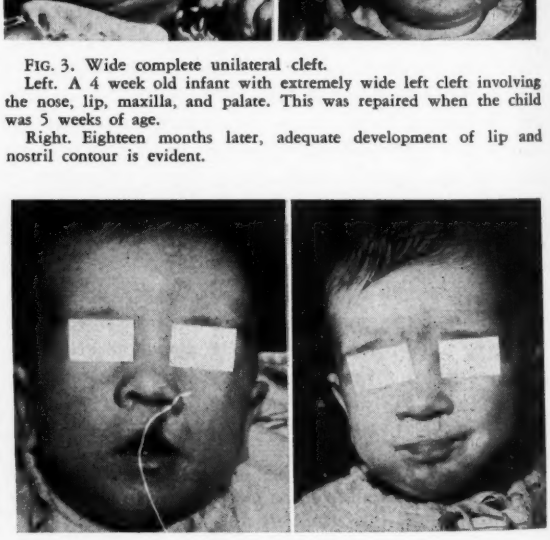

FIG. 4. Incomplete unilateral cleft.
Left. Incomplete left cleft of the lip only.
Right. Postoperative result photographed when the child was 8 months of age. Outline of the step-line scar is visible.

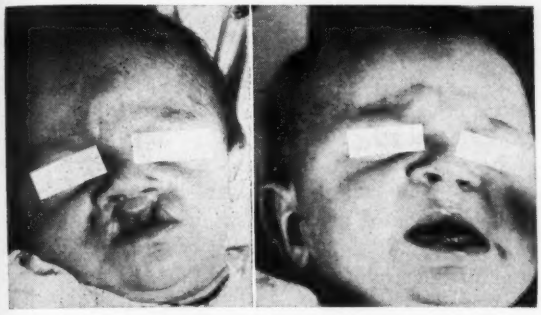

FIG. 5. Incomplete bilateral cleft.
Left. A 7 week old infant with incomplete bilateral cleft of the lip and alveolus.
Right. Postoperative view three weeks after a one-stage repair.

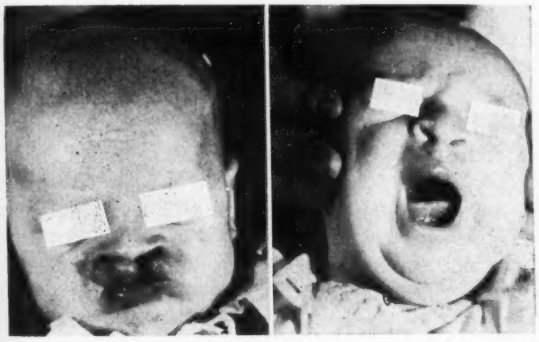

FIG. 6. Complete bilateral cleft.
Left. Preoperative view showing complete bilateral cleft with protruding premaxilla and prolabium.
Right. Two months after repair was completed in one operation. The displaced premaxilla was not manipulated but will return to a more nearly normal position because of pressure of the closed lip.

edges are denuded, and the square flap is then brought over to fit the reciprocal defect in the central part of the lip. Fine suture closure is accomplished in layers, including the mucosa, muscle, and skin. Suturing of the advanced mucosa in the buccal sulcus is important to produce the eversion or pouting of the lip. Closure in this manner results in complete muscle union without tension and a fine step-line scar which is slightly offset from the midline. A metal horseshoe-shaped apparatus, known as the Logan Bow, is applied to protect the wound and relieve the strain on the suture line.

POSTOPERATIVE CARE

After surgery, the infant is wrapped in a restraining jacket. Elixir of phenobarbital may be administered as indicated for sedation. The nursing staff cleanses the wound several times daily with a bland solution. Skin sutures and the Logan Bow are removed on the fifth or sixth postoperative day, and sutures in the mucous membrane in approximately two weeks.

Feeding is continued with the asepto-bulb syringe

FIG. 7. Secondary lip deformity in a 9 year old girl.
Left. Preoperative view showing a whistle deformity and grooved vertical line scar.
Right. Reconstructed lip using secondary repair. The lip contour was improved by a step-line closure and the vermillion border equalized. The nostril on the cleft side was elevated to the level of the opposite side.

or similar device until the wound is completely healed. Regular bottle nursing may then be instituted. Supportive vitamin and antibiotic therapy likewise is continued during hospitalization. Barring any unforseen complications the child is discharged from the hospital on the sixth to eighth postoperative day.

Follow-up care consists of close observation for possible cicatricial contracture or hypertrophic scar formation. Appropriate prophylactic measures include the application of a bland ointment and a minimal pressure dressing. Judicious use of irradiation therapy to prevent excessive scar formation sometimes may be indicated.

RESULTS

The LeMesurier procedure in my experience has produced gratifying results in both complete and incomplete unilateral clefts (fig. 1-4). Overall advantages of the repair are vertical-horizontal balance of the lip and improved profile contour. Specific values include a symmetrical alar level and nostril floor, a straight columella, a more nearly normal philtrum, and a full vermillion lip border. The operation also produces the desired cupid's bow with eversion or pouting and equalizes the thickness of the vermillion edge. Finally, the broken-line scar will not tend to contract or result in a notched "whistle" deformity of the vermillion border. With fine suture closure, the off-set step-line scar is barely noticeable and, above all, removes the classical harelip stigma of the straight-line repair.

As mentioned before, surgical correction of the double cleft is much more difficult than the unilateral cleft, and equally good results are not as consistent. Imperfections subsequent to a bilateral repair are usually the result of inadequate tissue to repair the extensive clefts. Figures 5 and 6 demonstrate respectively an incomplete and complete bilateral cleft repaired in a one-stage procedure.

Secondary revisions are often necessary to correct deformities remaining from the primary repair of those which develop during growth. A subsequent corrective procedure involves local readjustment of tissues and reconstruction by a similar method as outlined above for primary closure. A square flap operation designed to overcome depressed scars, whistle deformities, or inequality of the vermillion border has been described by Trusler and Glanz.[18] The secondary correction of a whistle deformity and grooved scar is illustrated in figure 7.

When the primary repair has resulted in extreme horizontal narrowing of the upper lip, a portion of lower lip may be transplanted in an operation known as the Stein-Estlander-Abbe lipswitch. Further measures sometimes indicated include a columellar lift to elevate a depressed nasal tip or a corrective rhinoplasty with cartilage or bony implants to the nose. The latter is more frequently necessary in patients who have congenital double clefts of the lip. Secondary revisions are done at different ages, depending upon the nature of the deformity. Usually, the lip repair takes precedence in younger patients, and nasoplastic procedures are best postponed until late adolescence.

CLEFT PALATE

Since clefts of the palate appear in association with cleft lips in approximately 50 per cent of these malformations,[19] mention should be made of their surgical correction. Closure of the cleft palate presents a challenging problem because of the more formidable procedure involved and the ultimate effect upon the child's speech. Defective speech in cleft palate patients is due to incomplete velopharyngeal closure during formation. Repair of the palatal deformity is advisable at 18 months of age and may be postponed if the child is not considered a good surgical risk. Palate procedures are more extensive than repairs of the lip, and the child of 18 months is better able to withstand the anesthesia and greater loss of blood. Also, the increased size of the head and mouth parts presents less technical difficulties than in a smaller infant. Closure of the palate before the child is 2 years of age is advocated, however, to meet the prime objective of good speech patterns. Surgery at this time also will overcome the susceptibility to chronic infections of the upper respiratory tract and occasional hearing loss subsequent to open palates.

Secondary repairs of residual palate perforations or shortened palates may be necessary as the child grows. It should be emphasized that surgery alone cannot accomplish the rehabilitation of patients with cleft palate. The combined efforts of the plastic surgeon, orthodontist, prosthodontist, and speech therapist are needed to obtain the rest results.

SUMMARY

Recent advances in the management of cleft lip deformities are presented with particular reference to the LeMesurier operation, which results in normal lip contour with a fine step-line scar and overcomes the stigma of the classical straight-line harelip closure. Preliminary care of the infant with cleft lip is explained, including the mechanics of feeding and other important preoperative considerations. The time for surgical correction of the lip is when the child is 4 to 6 weeks of age for unilateral clefts and slightly older for bilateral clefts, depending also on minimum weight requirements. The LeMesurier surgical technique is described briefly, followed by the significant details of postoperative care. It is noted that this procedure has been adapted for secondary revision of unsightly harelip scars. Cleft palate deformities, present in almost 50 per cent of infants with cleft lip, are discussed briefly. Closure of the palate is recommended at approximately 18 months of age.

Statistical information available in Texas on lip and palate malformations during the past five years gives an approximate incidence of 1 to 700 live births. Pertinent aspects of incidence, embryology and etiology of cleft lip deformities are included with a brief review of the literature.

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Lesions of the Esophagus in Infants and Children

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LESIONS of the esophagus in infants and children are uncommon, and yet their rarity should not provide an excuse for neglect or indifference because the majority are surgically curable.

Ingested foreign bodies which lodge in the esophagus and cicatrical changes in the esophagus after the ingestion of caustics are the most common abnormalities of the esophagus which the pediatrician and radiologist encounter. Accurate diagnosis in such cases offers no problem, for the history itself is diagnostic. However, other less common abnormalities, both acquired and congenital in origin, may present difficulties in diagnosis. It is the purpose of this report to call attention to these lesions and to describe their radiographic appearances.

LESIONS

Esophageal atresia with or without fistulous communication with the trachea constitutes the most commonly encountered congenital abnormality of the esophagus. In 1929 Vogt[8] grouped the various combinations of this disorder into a simple and usable classification (fig. 1). The most common type consists of a blind upper esophageal pouch with fistulous connection between the trachea or bronchus and distal esophageal segment (Vogt's type 3b).

Although clinically the symptoms of dysphagia, cyanosis, and choking attacks during attempted feedings in a newborn infant are characteristic of the anomaly, roentgenologic evaluation and confirmation are mandatory. The procedure is brief and nontraumatic to the infant. On the fluoroscopy table a small rubber catheter (no. 10-12 F) is inserted through either the nose or mouth and passed into the upper portion of the esophagus. Its exact location should be determined fluoroscopically not only to be certain that it has not entered the trachea but also to be sure that it has not coiled within the esophagus, thereby giving an erroneous impression as to the location of the obstruction (fig. 2a). If obstruction to passage of the tube down the esophagus occurs, a presumptive diagnosis of esophageal atresia may be made. With the patient in the prone or lateral position a single cubic centimeter of bland iodized oil is injected into the catheter. If atresia is present, the upper segment of the esophagus will be clearly outlined (fig. 2b and c). After roentgenograms are obtained, the opaque material is removed by suction and the catheter withdrawn. Aspiration of a small amount of the contrast substance into the trachea frequently occurs, but this in small amounts is innocuous.

If iodized oil is detected in the tracheobronchial tree, it may be difficult to determine if the substance was aspirated or if communication exists between the upper segment of the esophagus and the trachea. However, by judicious observation of the contrast substance as it first enters the esophagus with the patient in a lateral or oblique position, differentiation can be made.

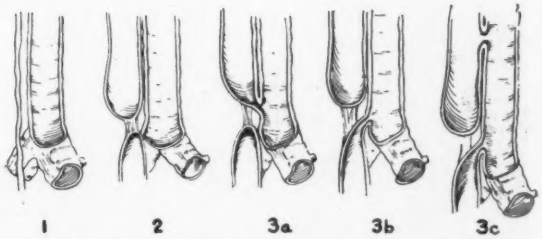

FIG. 1. Vogt's classification of esophageal anomalies. Type 3b is the most common.

The visualizing of gas within the gastrointestinal tract in the presence of esophageal atresia is substantial proof that there is communication between the tracheobronchial tree and the esophagus below the site of atresia. The absence of gas in the intestinal tract usually indicates the absence of a fistula (fig. 2d).

Rapid serial exposures of the esophagus during deglutition offers a reliable method of studying the function of the esophagus and the patency of the site of anastomosis after surgical repair. The majority of these follow-up examinations disclose lack of motility

From the Department of Roentgenology, Texas Children's Hospital.
Read before the Section on Pediatrics, Texas Medical Association, Annual Session, Houston, April 29, 1953.

of the esophagus, which probably is the result of interference with the autonomic innervation at the time of surgery; however, the possibility of congenital absence of the normal innervation should not be discounted. In any event the lack of motility does not appreciably handicap the patient. Figure 2e through h illustrates a case of historical interest in that in March, 1941, this patient underwent the first successful transthoracic repair of an esophageal atresia and tracheoesophageal fistula. The serial films made nine years afterward show the site of anastomosis as well as the lack of esophageal motility. The small traction diverticulum is probably the result of a primary tuberculous complex which the patient developed several months prior to the examination.

Tracheoesophageal fistula without atresia of the esophagus is a rare anomaly but should be considered in an infant with choking attacks during feeding. The fistula, which usually is small, frequently runs an oblique course caudally from the trachea to the esoph-

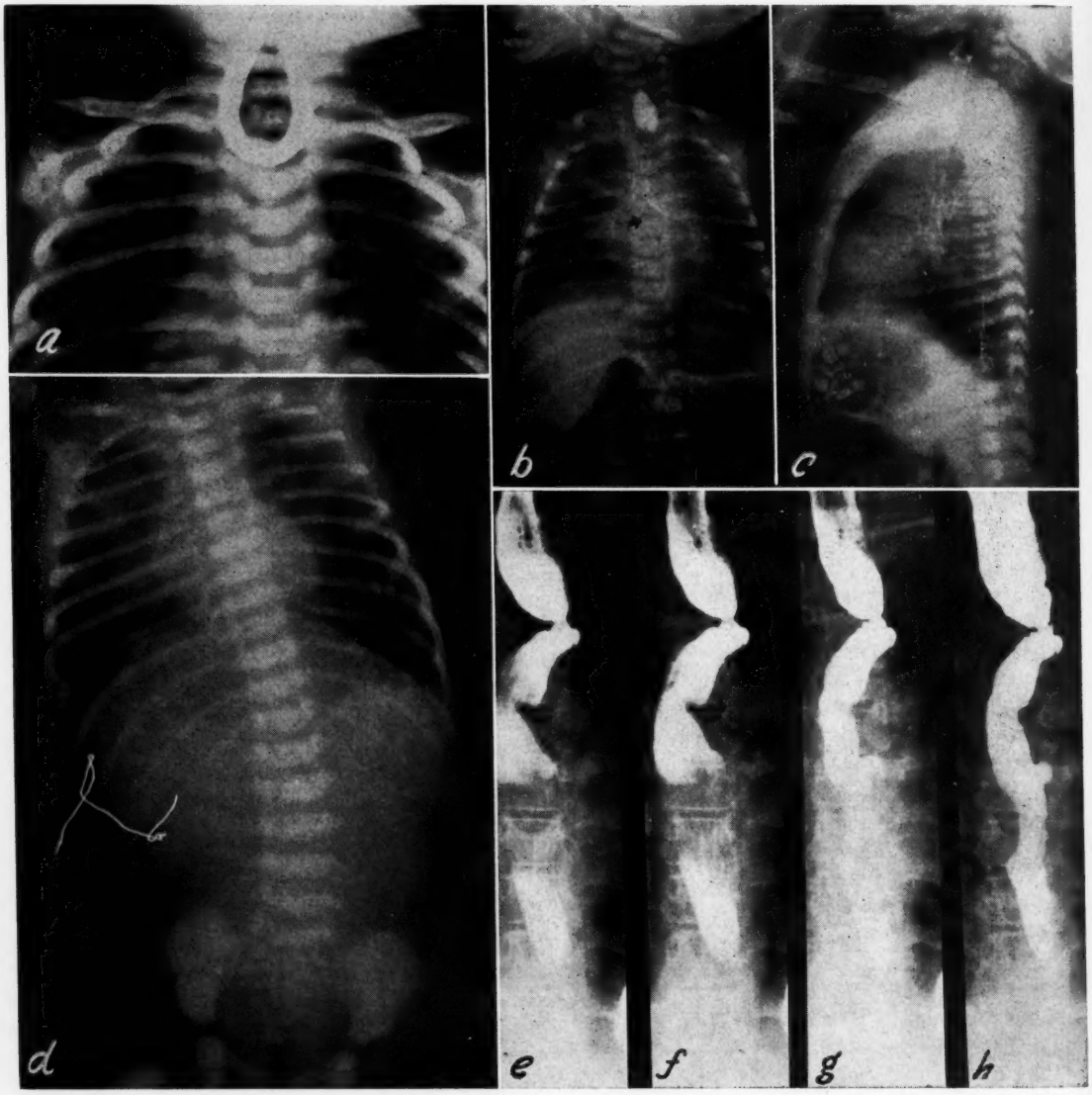

FIG. 2a. A rubber catheter coiled within the esophagus above the site of atresia indicates the necessity of radiography in accurately locating the level of obstruction.

b and c. Frontal and lateral views show esophageal atresia with fistulous communication between the left mainstem bronchus and the esophagus below the site of atresia. Aspiration of contrast material into the tracheobronchial tree led to visualization of the distal segment of the esophagus (*arrow*).

d. Lack of gas in the gastrointestinal tract in this patient with esophageal atresia is presumptive evidence that associated tracheoesophageal fistula is absent.

e through h. Serial films of the esophagus were made nine years after surgical repair of esophageal atresia and tracheoesophageal fistula. In spite of the apparently narrow anastomosis, dysphagia was not present.

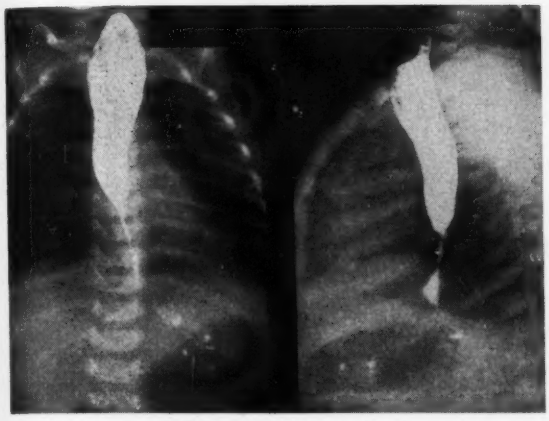

FIG. 3. Roentgenograms demonstrating probable congenital stenosis of the esophagus in a newborn infant.

agus and consequently may be demonstrable only with the infant in Trendelenburg position.

Congenital stenosis of the esophagus is extremely rare and often impossible to differentiate from an inflammatory stricture. The symptoms may be identical with esophageal atresia, and the necessity of using contrast material in the examination rather than simply noting the level at which obstruction to the rubber catheter occurs is obvious. Figure 3 represents a case of congenital stenosis of the esophagus in a newborn infant. Differentiation of this lesion from an inflammatory stricture secondary to peptic ulceration was not definitely determined. However, as a rule the latter condition affects the lower third of the esophagus, whereas the midportion is usually the site of congenital stenosis.

Duplications of the esophagus are anomalies which should be considered in the differentiation of intrathoracic masses in infants and children. According to Bremer,[1] esophageal duplication, as well as other duplication anomalies of the alimentary tract, represents the persistence of one of the hollow spaces formed by the vacuolation of the tubular mass of epithelial cells comprising the primitive alimentary tract. Esophageal duplication may produce classic symptoms and signs of an expanding intrathoracic lesion,[3] or the lesion may attain a large size before clinical findings become manifest. Chest roentgenograms show the abnormality posteriorly and therefore differentiation must be made from other posterior mediastinal tumors such as ganglioneuromas, neuroblastomas, neurofibromas, and anterior meningoceles. Because anomalies of the thoracic spine frequently are associated with esophageal duplication, differentiation from anterior meningocele may be impossible without myelography (fig. 4). Communication of the duplicated segment with the esophagus is seldom present. The cystic tumors are frequently lined with gastric mucosa, and consequently peptic ulceration and hemorrhages are not unusual complications.

Cardiospasm or achalasia of the esophagus is an acquired abnormality, the etiology of which is not definitely known. Overactivity of the sympathetic innervation of the lower part of the esophagus or inhibition of the parasympathetic nerves offers a logical explanation, but if and why such an imbalance occurs is unknown. Although adults are more commonly affected, the condition is not rare in children. A routine chest roentgenogram may suggest that achalasia exists, the dilated esophagus casting a band of increased density paralleling the spinal column. With barium in the

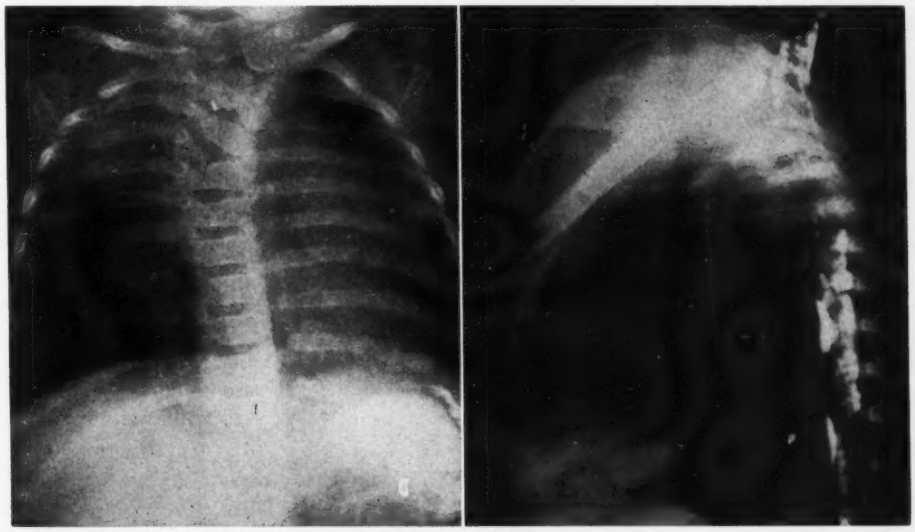

FIG. 4. Roentgenograms showing a posterior mediastinal mass. Note the anomalous development of the dorsal vertebrae, a frequent accompaniment of esophageal duplication. Myelography (*right*) ruled out suspected anterior meningocele.

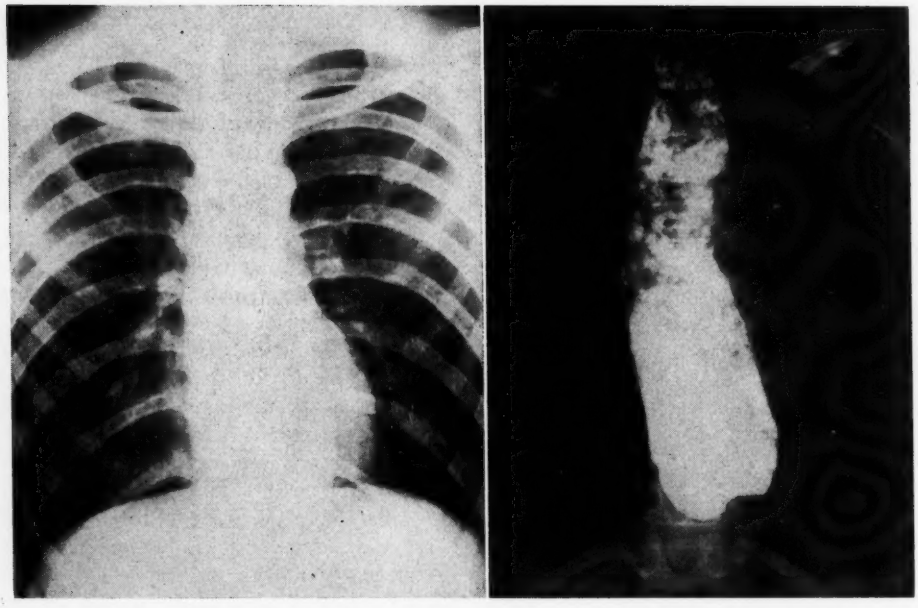

FIG. 5. Roentgenograms showing cardiospasm in a 10 year old boy. A dilated esophagus was seen on a routine chest roentgenogram (*left*). The esophagus was outlined with barium (*right*).

esophagus the appearance is diagnostic. The esophagus is dilated and often tortuous. The distal end is narrowed and allows at the most only a trickle of barium to pass into the stomach. The history as well as the abrupt change from dilated to stenotic esophagus differentiates the condition from caustic stricture. Roentgenograms of a 10 year old Negro boy who had a prolonged history of repeated vomiting attacks are shown in figure 5. The routine chest roentgenogram made prior to examination of the upper portion of the alimentary tract allowed a presumptive diagnosis of cardiospasm to be made.

Chalasia of the esophagus as one of the causes of persistent regurgitation of feedings in the newborn and young infant was first reported by Neuhauser[6] in 1946. In this condition there is persistent relaxation at the cardioesophageal junction, thereby allowing gastric contents to enter the esophagus. Whether this relaxation results from failure of function of the normal pinch-cock mechanism of the diaphragm because of faulty development or weakness of the diaphragmatic musculature, or to sympathetic-parasympathetic imbalance at the cardioesophageal junction is not known. Certainly the condition must be present to a mild degree in all newborn infants, as evidenced by fluoroscopic observation of the free movement of

FIG. 6. Roentgenogram illustrating cardioesophageal relaxation.

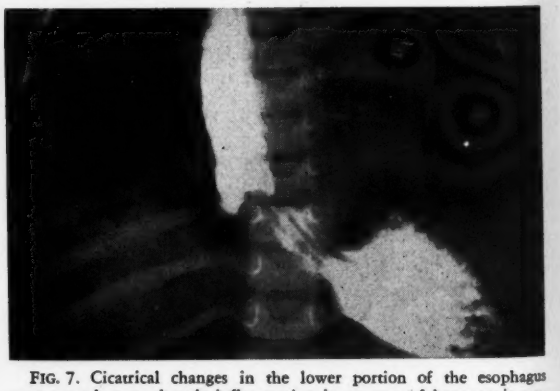

FIG. 7. Cicatrical changes in the lower portion of the esophagus were secondary to chronic inflammation in an area of heterotopic gastric mucosa. Note the resulting hiatus hernia.

contrast material from the stomach into the esophagus. The condition therefore probably should be considered as an exaggeration of the regurgitation which occurs in all infants rather than as a disease entity. In such cases contrast material is seen to enter the esophagus from the stomach when there is an increase in the intra-abdominal pressure either during inspiration or after pressure has been applied to the abdomen by the fluoroscopist's hand. As the infant becomes older, the frequency of vomiting becomes less and eventually, as in all babies, subsides entirely. The condition may be difficult or impossible to distinguish from a small hiatus hernia by roentgenologic examination. The patient whose roentgenogram is shown in figure 6 was a 6 month old Negro infant who had failed to gain satisfactorily since birth and who had regurgitated practically all of each feeding. During the fluoroscopic examination, the downward excursion of the diaphragm during inspiration resulted in propulsion of the contrast material from the stomach into the esophagus, promptly followed by regurgitation. Feeding in the upright position resulted in more adequate retention of the contrast material in the stomach.

Congenitally short esophagus with associated hiatus hernia as a diagnosis at one time was made rather frequently; however, the general concept now is that in most instances the esophagus becomes shortened as the result of peptic ulceration secondary to heterotopic gastric mucosa within the esophageal wall. The resulting fibrosis of the ulcerated site leads to contraction and shortening of the esophagus and eventually to traction displacement of the cardia through the esophageal hiatus. Microscopic evidence of gastric mucosa within the wall of the esophagus often provides proof for this assumption. However, other cases of short esophagus may result from esophageal ulceration secondary to a congenital hiatus hernia.[7] In these cases gastric juice reaches the esophagus through an incompetent esophageal hiatus and the chronicity of the process leads to ulceration. In view of this, one cannot help but wonder if the condition of chalasia of the esophagus described previously is not another predisposing factor in the development of esophageal ulceration and short esophagus.

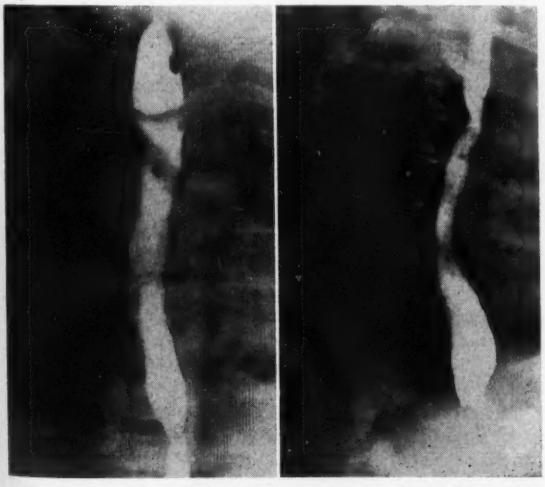

FIG. 8. An oblique filling defect on the posterior surface of the esophagus is seen in frontal (*left*) and oblique (*right*) views. Angiocardiography showed the right subclavian artery arising from the left side of the aortic arch and passing posteriorly to the esophagus to the right side of the neck. The diagnosis was confirmed at surgery.

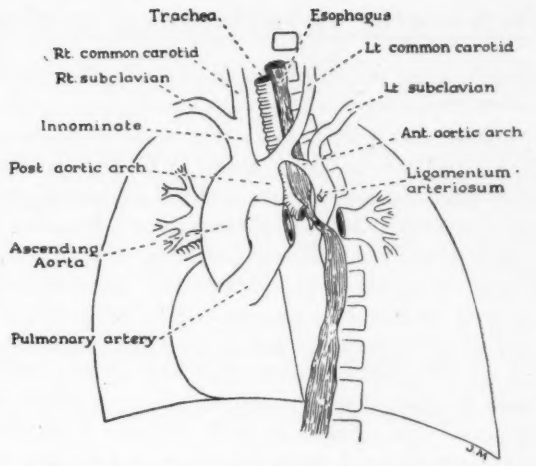

FIG. 9. A diagram illustrates double aortic arch with tracheal and esophageal compression (postoperative sketch).

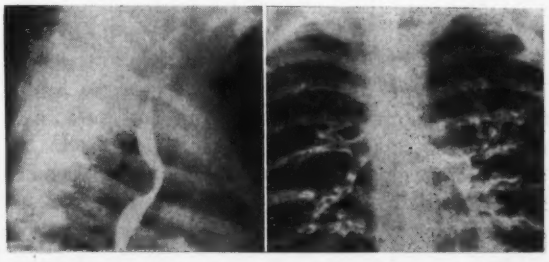

FIG. 10. A double aortic arch deformity with characteristic constrictive deformities of the esophagus and trachea is demonstrated by roentgenograms.

Dysphagia, retrosternal pain, and hematemesis are the most common symptoms of congenitally short esophagus.

By radiologic examination the lower portion of the esophagus is seen to be narrowed and ulceration frequently can be demonstrated. The esophagus also is shortened and the cardia of the stomach extends into the thorax. The hiatus hernia seen in figure 7 occurred in a 3 year old boy, and histologic examination made after resection of the stenotic segment of the lower part of the esophagus showed gastric mucosa. The pa-

tient had had frequent bouts of vomiting, the vomitus often containing blood.

Anomalous development of the vessels arising from the aortic arch frequently results in pressure deformities upon the esophagus of sufficient degree to produce dysphagia. Similar compression on the trachea is characterized by stridulous breathing. Vascular ring formation should be considered when either or both of these symptoms are present. Articles by Neuhauser[5] and Gross[2] describe in detail the various anomalies of the aortic arch, and they will not be dealt with here. However, I wish to call attention to two of the more common abnormalities in order to emphasize the importance of fluoroscopy and radiography in detecting as well as in accurately identifying the exact type of anomaly. The most common is the ectopic right subclavian artery. In such cases a normal aorta is present except that the right subclavian artery arises as the last vessel from the left side of the arch. In order to occupy its normal position in the right side of the neck, it must pass across the midline from left to right. The usual course is posterior to the esophagus, thereby producing a characteristic oblique defect on the posterior wall of the esophagus above the level of the aortic arch (fig. 8). The ectopic subclavian artery may pass between the esophagus and trachea, in which case the indentation will be on the anterior surface of the esophagus.

Double aortic arch, the second abnormality, results from persistence of the embryonal right portion of the fourth pair of aortic arches, which passes to the right and to the posterior aspect of the esophagus before joining the normally placed anterior limb to form a common descending aorta. The trachea and esophagus are thereby caught in a vascular ring which becomes more constrictive as the infant increases in size. Infants so affected may have alarming symptoms of respiratory distress often made worse after the ingestion of food. During inspiration marked substernal retraction may occur, and inspiratory and expiratory stridor are obvious. Figure 9 is a diagrammatic drawing of an operation on a 2 year old infant who had a double aortic arch. Preoperative studies of the esophagus and a tracheogram showed the characteristic constrictive deformity of a vascular ring (fig. 10).

SUMMARY

Although the types of lesions of the esophagus discussed in this paper are infrequently encountered outside large pediatric institutions, recognition by the pediatrician of the radiologist's ability to demonstrate the lesion and of the surgeon's ability to repair the defect is imperative for the welfare of the patient. Congenital lesions offer the most difficulty in diagnosis and hence are mainly considered.

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ABSTRACT OF DISCUSSION

DR. DENTON A. COOLEY, Houston: Recent developments in thoracic surgery have increased the interest of members of the medical profession in disorders of the esophagus, and modern esophageal surgery now depends upon and demands an accurate and precise diagnosis made possible by esophagoscopy and roentgenography. The barium swallow is the most valuable piece of evidence in determining the cause of dysphagia and dysfunctions of deglutition. More liberal use of the esophagram in conjunction with routine chest roentgenograms will reveal many latent and unsuspected lesions of the esophagus and provide opportunity for early correction.

Dr. Singleton has commented upon the radiologic findings associated with vascular anomalies of the aortic arch and its major tributaries. In cases of complete vascular ring, for example, double aortic arch or right sided arch with left ligamentum arteriosum, the symptoms of esophageal and particularly tracheal obstruction may be severe and lead to death in the first few months of life. Thus, the possibility of a vascular anomaly should be considered in any infant with respiratory stridor and the diagnosis confirmed by radiologic examination. Emergency thoracotomy and release of the strangulating ring may be lifesaving. Dysphagia lusoria has long been recognized as a symptom of esophageal obstruction produced by an aberrant right subclavian artery which in crossing the mediastinum may impinge upon the esophagus. In my experience with operations for pulmonic stenosis, the retroesophageal subclavian artery has been an incidental finding giving rise to no symptoms, and Blalock reports 26 such cases in which there was no dysphagia at all. The wall of the aberrant subclavian artery may, however, be thinner than normal, and I recently have seen an aneurysm of such a vessel in a 64 year old man who in spite of marked esophageal deviation had no symptoms. Such vessels also have been known to perforate into the esophagus with fatal hemorrhage. Such complications of aberrant vessels in the mediastinum possibly justify surgical exploration even in the absence of symptoms.

The early diagnosis of congenital atresia of the esophagus is imperative if surgical correction is to be successful. Unfortunately, infants still come to the surgeon many days after birth with disseminated pneumonitis caused by aspiration. If nursery personnel is alert, such cases can be discovered during the first twenty-four hours of life. The diagnosis is confirmed by radiologic examination, and immediate repair of the atresia probably will be successful.

ESOPHAGEAL LESIONS—Singleton—*continued*

Dr. Singleton has not dealt with acquired strictures of the esophagus from chemical burns. The importance of follow-up radiologic examinations of a burn to the esophagus caused by lye to avoid the insidious appearance of a stricture deserves emphasis. In some instances instrumentation and bougienage will prevent the development of a permanent stricture. Once a stricture is well established, however, esophageal resection with restoration of alimentary continuity by end-to-end anastomosis or esophagogastrostomy should be performed before the patient reaches an emaciated state, and a feeding gastrotomy should rarely be done. The current trend in such cases is toward early definitive surgery to spare the patient the morbidity and prolonged hospitalization of so-called conservative treatment.

DR. SINGLETON, closing: One additional word about patients who have respiratory difficulties: The physician should not consider the thymus as the responsible agent and use irradiation for these infants. He should look for another cause before blaming the thymus, placing abnormality of this structure at the bottom of the list as a cause for tracheal stridor.

Peroral Endoscopy in Cardiac Surgery

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WHEN one compares the symptomatology in cardiac disease with that in pulmonary disease, the similarity is at once apparent. The cough in pulmonary disease may be due to irritation of the sensory afferent endings in the bronchial mucosa by infection, malignancy, foreign body, or secretion. In cardiac disease it is due to a transudate of fluid in the terminal bronchioles which finally reaches a threshold of irritability, stimulating the cough to expel the material, or to pressure on the carina or main bronchi by enlarged vessels or an enlarged heart, as in cough due to compression of a bronchus by an enlarged lymph node.

Hemoptysis, that complaint which, fortunately, sends the patient hurrying to his doctor early, may result from actual ulceration, abnormal growth of friable tissue, erosion of a vessel, or infection of the bronchial mucosa.[7] In cardiac disease extravasation of the blood into the tracheobronchial tree will stimulate the cough, producing blood-streaked sputum. In mitral stenosis increased pulmonary hypertension with transudation of blood into the alveoli is the mechanism which produces hemoptysis. A true varicosity of the bronchial veins, reversing the anastomotic blood flow from pulmonary to bronchial veins because of increased venous pressure, is a specific etiologic factor. Hypertensive cardiovascular disease with left ventricular heart failure may produce a similar picture because of resultant increase in arterial pressure in the lesser circuit.[9]

Dyspnea, when caused by lung disease, generally denotes massive involvement of lung parenchyma. However, it can be elicited by any mechanism interfering with the mechanical, chemical, or nervous control of respiration. Anything which interferes with adequate oxygenation will produce dyspnea. Both acquired and congenital heart disease fit this category.

The wheeze which may be due to partial endobronchial obstruction from a tumor, foreign body, or extrinsic nodal compression is simulated in cardiac disease by compression of the bronchus or trachea by an aneurysm, anomalous vessel, or enlarged heart.

Certain observations of routine endoscopic examination not expected in any specific pulmonary disease should immediately bring to mind the possibility of cardiovascular disease. Paralysis of the left vocal cord should alert the examiner to the possibility of an enlarged left pulmonary artery which is pressing against the aorta and in turn pressing between them the left recurrent laryngeal nerve.[1, 2] Extrinsic compression of the trachea or bronchi also should bring to mind cardiovascular disease. I have noted a compression of the left main bronchus of varying degree in nearly every patient with cardiac enlargement of 2 plus or more in whom bronchoscopy is done for postoperative secretions. I have not observed any appreciable increase in the amount of retained secretions in the left bronchus as compared with the right in spite of the fact that one would surmise it would be greater. This compression and distortion often prevents passage of the bronchoscope into the bronchus to the left lower lobe, and aspiration should be done blindly with a flexible tip rather than traumatizing the mucosa by forcing. Of course, the trachea will be compressed in varying degrees by an aberrant innominate artery or a double aortic arch. Not infrequently engorgement of the small mucosal vessels in the bronchi will be visualized with the bronchoscope. Other than these few exceptions, however, examination of the tracheobronchial tree would correspond to that of any routine examination and the observations would be those of specific pulmonary disease.

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Investigation of the pulmonary complaints, mentioned previously as being so similar, will reveal anatomic conditions which, when properly correlated with the clinical and other investigative studies, will aid materially in establishing a specific diagnosis. Esophagoscopy in the patient with dysphagia caused by cardiovascular disease will show a compression of the esophageal lumen; this in turn may result from either an aberrant right subclavian artery or a double aortic arch.[3, 10] Compression of this subclavian artery with the tip of the esophagoscope will result in a loss of the radial pulse in the right wrist, with a normal pulse in the left wrist.[15] Although it is not necessary to do diagnostic esophagoscopy in every case of suspected vascular anomaly, the phenomenon described, when observed in a patient in whom such an anomaly is not suspected, will aid in channeling diagnostic efforts in the right direction.

Diagnostic bronchoscopy is indicated and should be carried out for those cases of cardiac disease in which there is a reasonable suspicion of associated pulmonary disease[8] as evidenced by segmental atelectasis, a questionable pulmonary infarction, diffuse basilar fibrosis with productive cough, or suspicion of tuberculosis based on roentgenographic evidence. After cardiac surgery, retained secretions make it imperative that bronchoscopy be performed.

The indications for bronchoscopy in the postoperative period are not nearly so clearly defined as the textbooks would have one believe. It should be the physician's aim to perform bronchoscopic examination before the classic physical and roentgenographic evidence of atelectasis is noted; in this sense it is a preventive procedure. In these cases the usual picture is that of a patient with a feeble cough, slightly diminished breath sounds on one side, and perhaps an elevation of temperature; nearly always there is no roentgenographic evidence of atelectasis. In some cases an isolated instance of temperature elevation or unexplained dyspnea, or an absence of breath sounds will be considered enough of an indication for what I call a "rule-out bronchoscopy." In these cases of cardiac surgery every effort should be made to avoid any unnecessary complication or prolongation of a complication. To achieve this end, bronchoscopy should be resorted to without hesitation and without undue risk. The finding of a classic picture of postoperative atelectasis should be rare and, if seen, might well indicate the patient had not received the careful attention needed to prevent it.

Of importance, therefore, is the actual technique and rationale of endoscopy in the patient with abnormal cardiac musculature. Immediately, the examiner's first thought is how much additional strain the heart will tolerate. I think I am safe in saying it will take a lot, providing certain precautions are observed.

PRECAUTIONS

The first problem to be met concerns premedication. In patients with cardiac disease Demerol should replace the usual endoscopic premedication of morphine because it does not depress the respiration or cough. I have noted that its sedative and euphoric action is equal to morphine, and it does not increase the tone of the vagus nerve as much as morphine. The use of atropine sulfate as a preoperative bronchoscopic medication raises no question. The problem of thickening and drying out the already viscid secretion in the postoperative patient is to be considered but is of less importance than the awareness that a diseased myocardium is as vulnerable to vagal stimulation under local anesthesia as a normal myocardium is under general anesthesia.[11] Such reflexes, of course, can be initiated from any of the nerve endings in the bronchial, vascular, or pleural group of vagus nerve ending.[13] Therefore, I suggest the use of atropine sulfate in appropriate dosage (grains 1/200 to grains 1/150) for bronchoscopy, both preoperatively and postoperatively in the patient with cardiac disease.[12] Finally, Nembutal is used to supplement the local anesthetic; my choice of anesthetic is cocaine.

Because most patients are dyspneic and to lie flat may be something they have not done for years, bronchoscopy is performed with the patient in bed lying in a semielevated position. The head of the bed is raised enough to allow the endoscopist to stand behind it, either on a bedside stool or on the crossbars. A pillow is placed behind the patient's shoulders, allowing the head to be extended. The bronchoscope may be introduced with or without the laryngoscope. Local anesthesia is similar to that for routine bronchoscopy in any diagnostic case, with the exception that instead of 10 per cent cocaine I have been using 2 cc. of 4 per cent cocaine instilled fractionally into the tracheobronchial tree and 4 per cent cocaine as a spray to the pharynx with satisfactory results.[5, 6, 14]

I think it is important to use oxygen as a supportive measure during the entire procedure, insufflating it through the oxygen channel on the bronchoscope. In addition, the patient should receive oxygen by mask or nasal catheter both before and after bronchoscopy, especially while traveling to and from the operating room if such a move is necessary.

Additional precautions to be taken in doing endoscopy involve the provision of equipment which theoretically should be available in every well equipped operating room but which is often overlooked for endoscopy. It should be understood that endoscopy in a patient with cardiac disease should not be carried out unless such equipment is available. Specifically, I refer

to a thoracotomy tray containing the necessary instruments for an emergency opening of the chest for cardiac massage. A defibrillation unit should be available. Of course, resuscitative drugs with appropriate means of instillation should be at hand.[4] Endotracheal catheters, which should be not used if possible because of further activation of vagal stimuli, should be present along with means of providing positive pressure to assist respiration when the chest is open. Needless to say, the immediate availability of a competent anesthesiologist is extremely desirable. However, for a few minutes the endoscopist in an emergency may act in the capacity of thoracic surgeon, anesthesiologist, cardiologist, and internist until more specialized consultants can be obtained. The patient with cardiac arrest will not wait for the elevator.

In infants and small children tracheal aspiration using a laryngoscope and flexible catheter is of value in the immediate postoperative period. It is a procedure which can be carried out with a minimum of risk by the less skilled attendant, and bronchoscopy can be reserved for the patient who does not respond to this therapy. Bronchoscopy repeated at frequent intervals in an infant usually results in the need for tracheotomy,[5] a procedure to be avoided if possible because of the associated pulmonary complications but to be done without hesitation if indicated. Aspiration by catheter performed by nurses, via the nares, with introduction of the catheter blindly into the trachea is of value in stimulating the patient to cough as well as in aspirating secretions which otherwise would cause obstruction.

Tracheotomy in the adult should be resorted to quickly when retained secretions in a patient in critical condition complicate an operative problem which is already precarious. This is particularly true in the patient with postoperative pulmonary bleeding or edema. In cases of cerebral embolus and coma, a tracheotomy should be done before pulmonary complications arise and is almost a routine procedure.[6]

In those patients who are considered inoperable, aspiration by catheter of accumulated secretion often will ease the burden on an already failing heart. Oxygen bubbled through alcohol has been observed to be effective in pulmonary edema. The Pneophore machine with positive pressure assistance is also of value in these cases.

SUMMARY

I would like to restate the position of physicians in the very specialized field of peroral endoscopy for cardiac surgery. As diagnosticians they should be more aware of cardiac disease as an etiologic factor when confronted with so-called "typical pulmonary complaints." They should make easily available safe, efficient, and reliable endoscopy so that the patient with cardiac disease may have the benefit of endoscopy when indicated. Lastly, they should realize that bronchoscopy after cardiac surgery has the same indications as for any other thoracic condition. There is no undue risk, and if reasonable precautions are taken, the procedure can be considered safe and resorted to without hesitation.

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ABSTRACT OF DISCUSSION

DR. ROBERT E. PARRISH, San Antonio: Dr. Maloney has compared the symptomatology in cardiac disease with that in pulmonary disease. He has shown that a number of conditions produce symptoms so closely similar that peroral endoscopy may be necessary to differentiate or aid in the differentiation of their true etiology.

Anyone doing peroral endoscopy has a great responsibility; the patient's welfare depends on the care and accuracy of the endoscopist. It is necessary for competent diagnosis and treatment that the peroral endoscopist have a fair knowledge of anesthesiology, internal medicine, and general chest surgery. He also should be able to read roentgenograms of chest lesions, a skill which is a valuable aid in the management of these patients. As mentioned in Dr. Maloney's paper, the endoscopist often is compelled to act in haste to overcome emergencies which compel a broad knowledge of medicine. In my opinion, the basic training of the laryngologist best fits a physician to acquire the specialized training necessary in peroral endoscopy.

Bronchoscopy is often needed after surgery and it is especially beneficial after surgery on the lung or heart. Dr. Ma-

loney emphasizes the necessity for bronchoscopy very early before danger signals arise; he uses it to prevent complications. No doubt physicians often have been reluctant to perform bronchoscopy before symptoms, signs, or roentgenographic observations indicate trouble.

Twenty-eight years ago I did my first bedside bronchoscopy in an attempt to relieve a patient with pulmonary edema after extensive abdominal surgery. The trachea and bronchi were aspirated after the pharynx and larynx had been sprayed with a small amount of 4 per cent cocaine. After aspiration, the patient began to improve and recovered.

In many conditions, if the cough reflex is not adequate to remove secretions from the respiratory tract, it is necessary to aspirate them either by bronchoscopy or catheteral suction after tracheotomy. If it is anticipated that excessive secretions may recur for a considerable period, I believe tracheotomy is better and safer; frequent aspiration can be done without the specialist's constant presence.

Dr. Maloney has covered many details pertaining to bronchoscopy as it may be performed for many different conditions. He mentioned only cocaine as the local anesthetic; others use newer anesthetics, especially Pontocaine hydrochloride. He uses atropine along with Demerol. In routine bronchoscopy I prefer not to use atropine. No doubt in cardiac work it is to be preferred. I have not had sufficient experience with patients having cardiac disease to determine the medication of choice.

The reader no doubt is impressed with the care Dr. Maloney takes when doing bedside bronchoscopy. The setup available to him is about the same as he would have in the operating room. The value is great, because movement of the patient is not necessary and frequent attention is possible. Constant watchfulness of the patient, with the necessary equipment available at all times, pays off in a lower mortality rate.

PULMONARY CYSTS

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THE increasing frequency of reports of pulmonary cysts in the literature may be attributed to (1) a wider use of roentgen examinations not only as a diagnostic procedure but as a measure of the result of treatment of pulmonary disease and (2) the rapid development of thoracic surgery, which has broadened the therapeutic approach to pulmonary pathologic processes and made a more accurate differential diagnosis necessary.

A suitable classification of lung cysts has not been agreed upon. It is undoubtedly of great academic importance to determine whether a cyst is congenital or acquired or whether it originates in a bronchus or in the alveolar structures, but neither the etiology nor the location can always be determined clinically. It is now believed by many that pathologists do not have accurate criteria for the differential diagnosis between congenital and acquired lung cysts, nor can they always say whether the lesion originated in the bronchus or alveolar structures.

In this paper the term "pulmonary cyst" is used to indicate all fluid or air filled sacs that occur within the lung tissue except tuberculous cavities, cysts caused by parasitic infestations, and excavations occurring within neoplasms. An effort is made to discuss the clinical features that lead to the early recognition of pulmonary cysts, the usual clinical courses that they run, and the best methods of treatment. A workable clinical classification which has proved helpful to us is as follows: (1) congenital or developmental lung cysts, (2) cystic cavities discovered in association with acute infections in the lungs of infants and young children, (3) cysts developing in certain volatile oil pneumonias, and (4) emphysematous blebs or bullae which usually occur in the aged.

Admittedly, this is only a rough classification, but it is useful in the clinical management of patients, since there is a marked variation in the behavior of cases in these different groups. We have selected representative cases illustrating the course taken by the various types of cysts.

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**University of Tennessee School of Medicine, Department of Roentgenology, and Methodist Hospital.*

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DEVELOPMENTAL CYSTS

CASE 1.—I. L. W., a white woman, aged 20 years, was admitted to the hospital October 11, 1950. Pulmonary symptoms had begun eight years before with cough, sputum, fever, and hemoptysis after an attack of measles. A diagnosis of lung abscess was made. The patient recovered from this episode and had had no symptoms until the present illness, which had started three weeks prior to admission of the patient. At that time a rapid onset of severe pulmonary symptoms occurred. A chest radiograph (fig. 1a) revealed a fairly sharply circumscribed homogeneous pneumonic-like process occupying the lower half of the right hemithorax with a suggestion of displacement of the heart shadow toward the left. The symptoms promptly subsided after chemotherapy, but after several weeks of conservative treatment a chest radiograph still showed residual disease (fig. 1b). Continuation of conservative treatment for a total of six weeks resulted in no further change in the roentgenographic appearance.

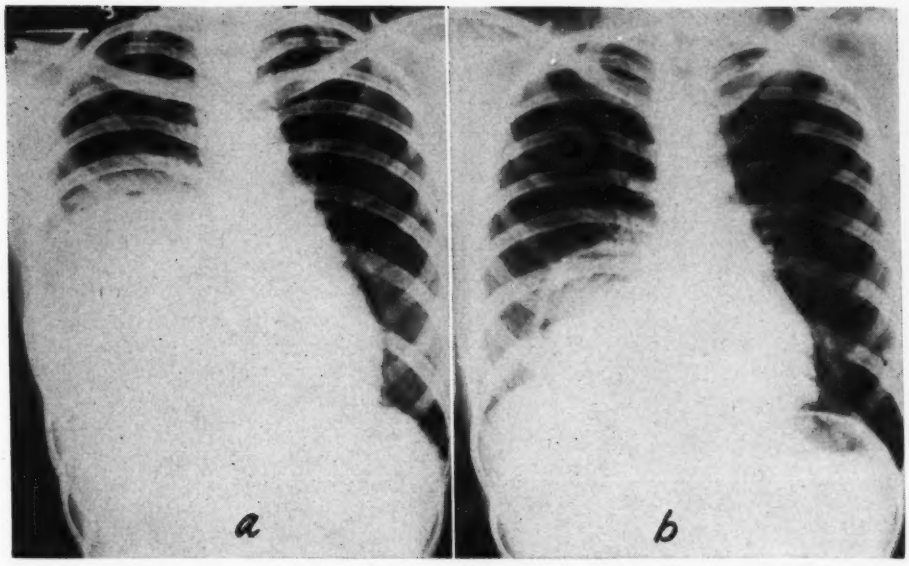

FIG. 1. Case 1. a. A pneumonic-like density in the lower half of the right hemithorax with a suggestion of displacement of the heart shadow toward the left.

b. Case 1. After maximum response to antibiotics, the residual lesion in the right base is obviously an infected cyst, but the patient was asymptomatic.

Exploratory thoracotomy demonstrated a cystic mass in the lower lobe of the right lung. An anomalous systemic artery arising from the aorta entered the lower lobe at the level of the cyst. Gross and microscopic examination showed the cyst to have several bronchial communications, and the anomalous systemic artery anastomosed freely with the normal pulmonary circulation.

CASE 2.—M. R. B., a 42 year old white woman, was admitted to the hospital March 16, 1949. In a routine roentgenographic survey of school teachers a shadow was seen in the right side of her chest (fig. 2a). There was a history of pleurisy on the right side fourteen years prior to discovery of the lesion in the lung.

At exploratory thoracotomy five days after admission a cystic tumor was found within the substance of the lower lobe of the right lung. An anomalous artery 1.5 cm. in diameter arose from the aorta and entered the lobe in the vicinity of the cyst. On examination of the surgical specimen a cyst 6 cm. in diameter was observed in the lobe. It was lined with bronchial epithelium and filled with a gelatinous mucoid material. The anomalous artery was injected and appeared to supply only the cystic area of the lobe.

These 2 cases present extremes of clinical findings. In the first patient there was a long history of recurrent episodes of respiratory symptoms, and a cyst in the lung communicated freely with the bronchial tree and acted as a reservoir of infectious material. In the second case little or no significant illness could be attributed to the cyst, which was isolated from the remainder of the bronchial tree and had no evidence of infection. However, in both cases a developmental defect in early fetal life almost certainly was responsible

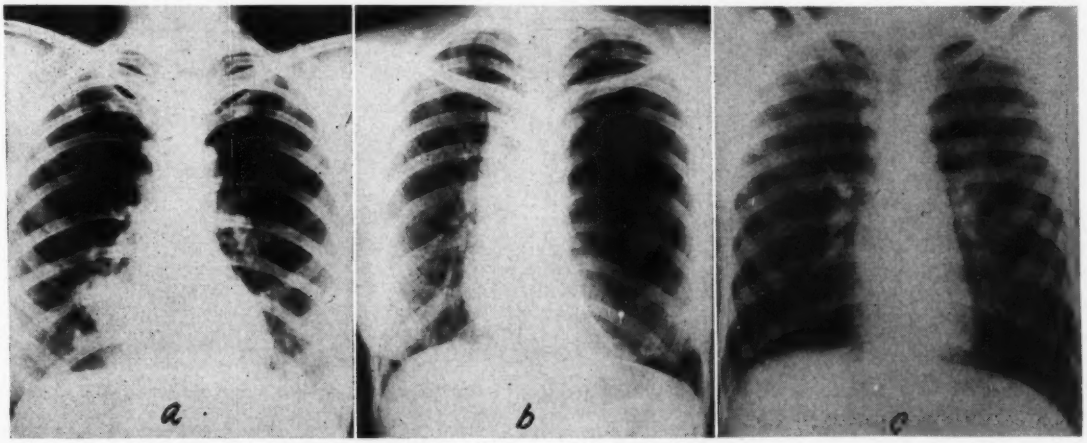

FIG. 2a. Case 2. A sharply delineated homogeneous density observed in a chest survey in a clinically well patient.

b. Case 3. An expansile air cyst occupying almost all of the left hemithorax and displacing the mediastinum to the right.

c. Case 4. A multilocular cyst located in the anterior basal segment of the lower lobe of the right lung.

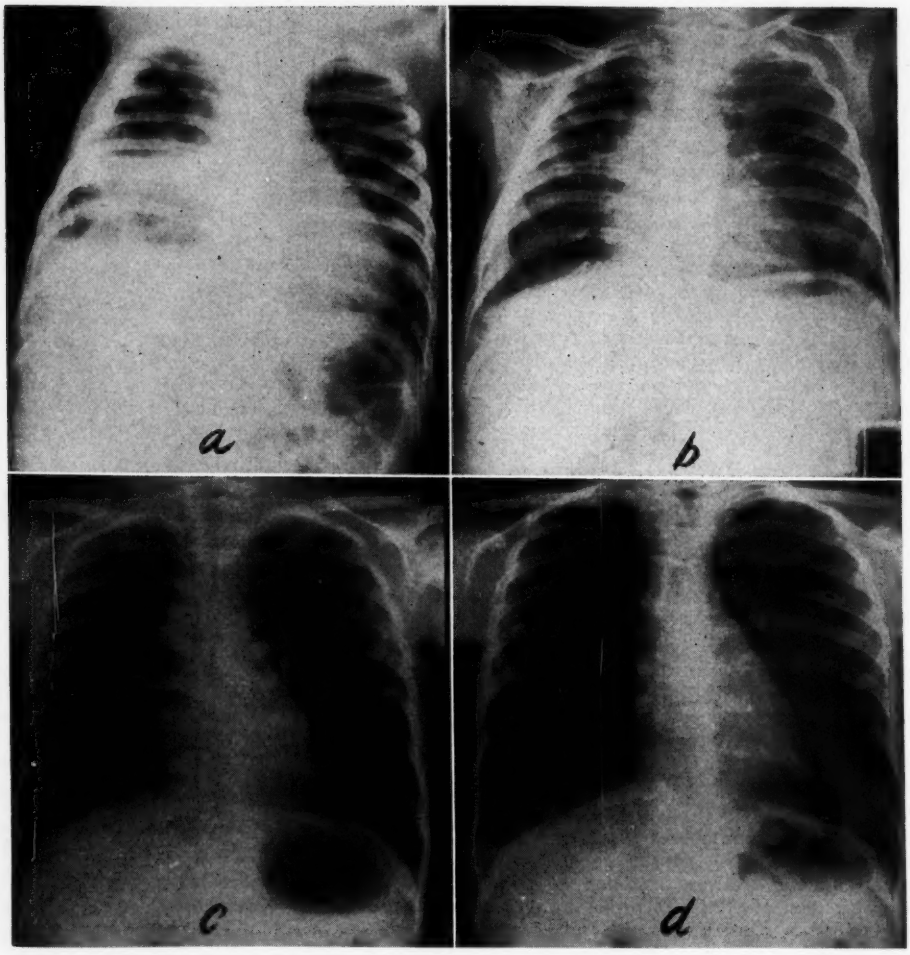

FIG. 3. Case 5. a. In this roentgenogram the right lung is replaced by cystic sacs and atelectasis or pneumonia.
b. Improvement is evident after seven weeks of conservative therapy.
c and d. A residual strandlike density in the upper right lung field is apparent in follow-up roentgenograms four and five years after the initial onset of disease.

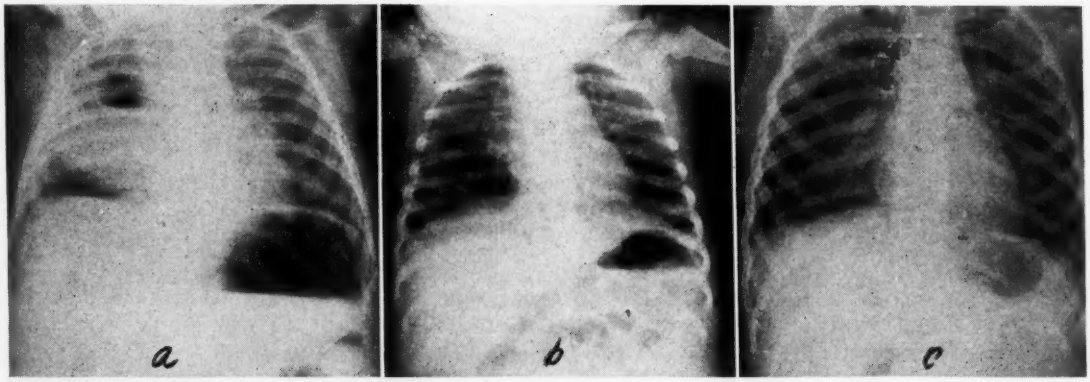

FIG. 4. Case 6. a. A homogeneous density with apparent fluid level is evident in the right midlung field. Note the absence of pleural reaction in the roentgenogram.
b. After six weeks of medical management, the cyst is visible but the infection has cleared.
c. Twenty-five months after onset of symptoms, the chest appears normal.

for the cyst. In each case an anomalous systemic artery entered the lung; these systemic arteries normally disappear early in fetal life. It is important that the chest surgeon remember the possibility of an anomalous systemic artery entering the lung, as disastrous hemorrhage could result from its inadvertent division. Those below the root region always have been seen in association with cystic changes in the lung or congenital defects of the diaphragm.[4]

CASE 3.—L. S., a 28 year old white woman, was admitted to the hospital because of recurrent respiratory infections. The present episode of cough, low grade fever, and dyspnea had persisted for one month. The patient had experienced intermittent chest pain and had been short of breath as long as she could remember. She never had been able to carry on the physical exertion normal for her age. Roentgen examination (fig. 2b) revealed a large cyst filling the entire left side of the chest and displacing the heart and mediastinum to the right.

At exploratory operation the entire hemithorax was filled with a cyst which involved most of the lower lobe of the left lung; the upper lobe was compressed against the superior mediastinum. The lower lobe with its cyst was removed in the usual fashion. Microscopic study of the resected specimen showed the wall of the cyst to contain cartilage, bronchial glands, and bronchial epithelium. Postoperative recovery was uneventful, and the upper lobe expanded to fill the chest. In the five years since operation, the patient has remained free from significant respiratory disease, and her general health is now normal.

The microscopic examination revealing all of the normal constituents of the bronchial wall within the wall of the cyst, plus the history of dyspnea all of the patient's life, seemed to justify the classification of this lesion as developmental.

CASE 4.—W. G. L., a 14 year old white boy, was admitted to the hospital for study because of recurring episodes of pulmonary infection since he had an attack of pneumonia at the age of 4 years. Pneumonia had been diagnosed again when he was 6 years of age. Productive cough always had been present.

The child was underdeveloped and had not been able to tolerate the physical exertion normal for his age. A chest radiograph (fig. 2c) revealed what appeared to be a cyst in the anterior basal segment of the lower lobe of the right lung. A bronchogram revealed bronchiectatic dilatations adjacent to the cyst. As is usual, the cyst itself had failed to fill.

Exploratory thoracotomy revealed extensive adhesions over the entire surface of the right lung. The lower lobe was resected. Several multilocular cysts were present. The adjacent pulmonary tissue was atelectatic and contained dilated bronchi. On microscopic examination the cysts were lined with ciliated respiratory epithelium which was so well defined that a developmental basis for the cystic disease was considered likely. In the fifteen months since operation, the child has been much improved in general health, and he now appears normal.

CYSTS WITH INFECTION

The next 3 cases represent cystic processes developing upon or in the presence of pneumonic lesions. This type of cyst appears to be peculiar to infants and very young children. In view of the present concept that the period of lung development begins at about the third week of prenatal life and extends to the fifteenth or sixteenth year of postnatal life,[2, 7] the question arises as to whether or not this type of cyst is developmental and, therefore, in many respects similar to congenital cysts.

CASE 5.—J. R., a white girl, aged 3 months, was admitted to the hospital with a ten day history of fever and cough. The symptoms had progressively increased, and at admission the child was seriously ill. A chest radiograph (fig. 3a) showed what appeared to be multilocular cysts in both the upper and lower lung fields on the right with atelectasis or pneumonia of the remaining lung tissue on this side. After supportive treatment, restoration of fluid balance, and administration of antibiotic drugs, the child improved.

Seven weeks after the beginning of treatment (fig. 3b) the lung fields had cleared of the pneumonic-like process, and there was noticeable reduction in the size of the cysts. Serial roentgen studies during the following year showed a further reduction in size, and there were no episodes of pulmonary infection. Follow-up examinations four and five years after the initial illness showed the child to have remained well clinically and to have developed normally. Roentgenograms (fig. 3c and d) showed only the faintest abnormality consisting of a strandlike increase in density just above the hilus with overdistended lung tissue in the right apex.

CASE 6.—M. H., a white girl, was admitted to the hospital at the age of 4 months with the history of pneumonia one month prior to entry. After treatment the acute symptoms subsided, but cough and fever in the afternoons persisted. Roentgen examination (fig. 4a) showed a large homogeneous density in the posterior part of the right midlung field containing a cavity with fluid level. There was some compression of the adjacent pulmonary tissue but little or no pleural reaction.

Within seven days after treatment with antibiotic agents and blood transfusion, the temperature returned to normal. Repeated roentgen studies showed a reduction in the size of the cyst and evacuation of its fluid contents within eight days. Six weeks later (fig. 4b) the cyst was still present but contained no fluid, and the right lung and hemithorax appeared normal in size. Without further episodes of respiratory infections the child has progressed normally, and a roentgenogram made twenty-five months after the initial illness was essentially normal (fig. 4c).

CASE 7.—C. A. was admitted to the hospital at the age of 3½ months after three days of dyspnea and fever. On roentgen examination (fig. 5a) there was seen a homogeneous density in the lower lobe of the right lung containing in its upper part a cavity with a fluid level. On treatment with antibiotic drugs and supportive measures the temperature promptly returned to normal. There was a rapid decrease in the size of the cyst and clearing of the adjacent pulmonary density. Check-up examination twenty-four months after the initial illness (fig. 5b) revealed normal observations. The child is in good health and has developed normally.

It is obvious from cases 5, 6, and 7 that this type of cyst can only be suspected or detected by serial radiographic examination, the most important study being a radiograph of the chest after the child appears well clinically. Such studies are obviously the most accurate means of determining the complete clearing

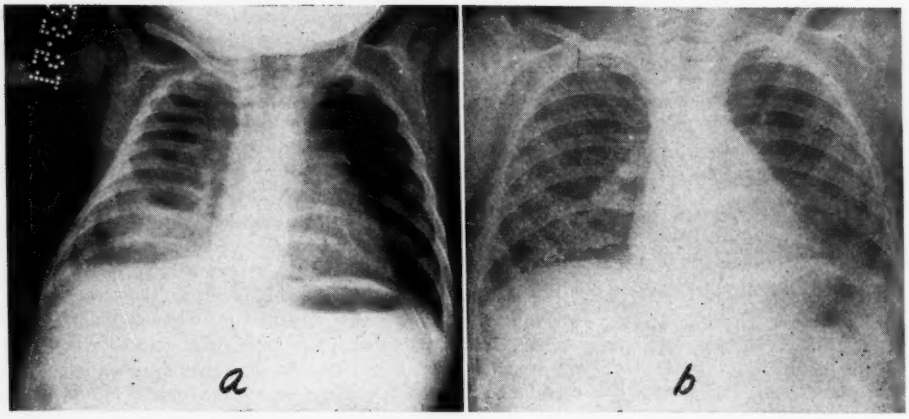

FIG. 5. Case 7. a. A homogeneous density in the lower lobe of the right lung contains evidence in its upper part of a cavity with a fluid level.

b. Twenty-four months after the initial illness, the check-up roentgenogram appears to be normal.

of chest disease. Caffey[3] recently reported 13 cases of pulmonary cyst in infants that regressed without surgical treatment. Although most cysts that complicate pneumonic lesions in infants clear under conservative therapy, the rapidly expanding cysts that produce tension and result in death by asphyxia are occasionally seen in this group. It is necessary to observe the patient closely in order that respiratory emergencies can be met promptly by proper therapeutic procedures.

CYSTS FROM VOLATILE OILS

The literature contains numerous reports[5, 8] of pulmonary complications after the inhalation or ingestion of volatile oils. In the following case aspiration of

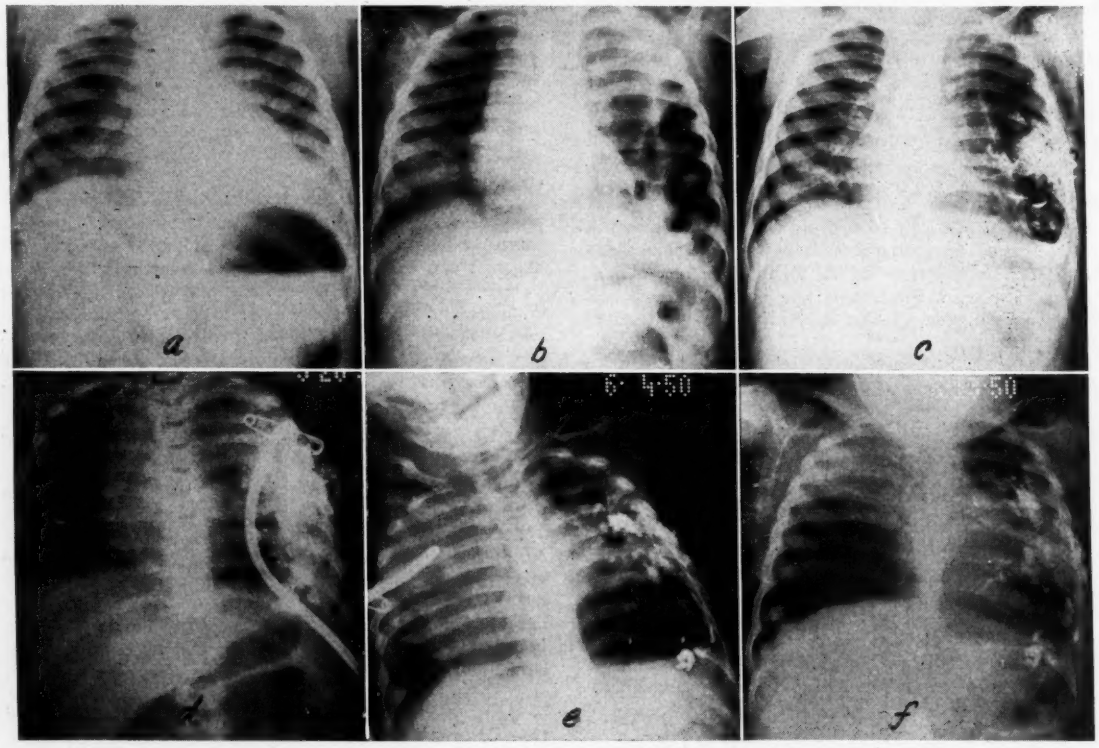

FIG. 6. Case 8. a. Residual kerosene pneumonia, bilaterally.

b. Eight days later, cysts are evident in the left base with pneumothorax and a cyst appears in the right lower lung field.

c. One week later there is increased pneumothorax on the left; a contrast medium is in the pleural space and in the lung cysts. Note the subcutaneous emphysema.

d. Decompression of the left side. The cyst on the right has increased.

e. Two weeks later, the right side is decompressed. The left lung has expanded, but the cysts remain bilaterally.

f. Nine days later, both lungs are expanded. The child is improving.

kerosene was followed by bilateral cysts of the lung with resulting severe pulmonary complications.

CASE 8.—E. B., an 18 month old white girl, was admitted to the hospital with fever, cough, and lassitude. She had aspirated a small amount of kerosene twelve days before. Immediately after the accident the stomach was lavaged. One week later pneumonia developed; the patient was hospitalized and given penicillin. The symptoms improved, but a chest radiograph (fig. 6a) showed residual disease in both lower lung fields, and the child was transferred for hospitalization under a specialist's care. Despite antibiotic therapy and supportive measures, she did not improve, and eight days after the first roentgenogram, a chest radiograph (fig. 6b) revealed loculated pneumothorax in the left base with cysts in the adjacent lung tissue. On the right side in the region of the middle lobe there was an area of homogeneous density with a small cavity in its upper portion. The clinical condition deteriorated, fever increased, and dyspnea progressed to the point of cyanosis. Attempts to decompress the air spaces on the left by thoracentesis gave temporary relief but resulted in extensive subcutaneous emphysema, which further increased the patient's discomfort. Further decompression was carried out (fig. 6c) and iodized oil was injected; the oil outlined the cysts within the lung as well as air in the pleural space. Meanwhile, the cyst on the right side progressively increased in size. Eventually bilateral tension pneumothorax occurred, and aspiration and drainage by catheter of both pleural spaces was necessary (fig. 6d and e).

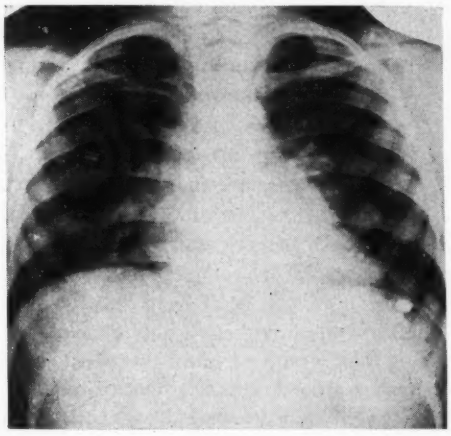

FIG. 7. Case 8. Thirty-four months after onset of residual kerosene pneumonia. No cysts are visible, but evidence of chronic pulmonary disease persists.

After approximately six weeks of a stormy clinical course the child began to improve. A chest radiograph (fig. 6f) showed the cysts to be much smaller. There was more expansion of adjacent lung tissue, and the pneumothorax spaces had disappeared. Continued improvement followed, and the child was dismissed four weeks later, at which time there was considerable residual density bilaterally with cysts present in both bases.

Follow-up examination in the outpatient department showed a gradual improvement in the child's condition. Gain in weight and growth were normal. However, thirty-four months after admission the child still was having continuous colds accompanied by persistent cough. Roentgen examination at this time (fig. 7) showed evidence of chronic disease in the lower lung fields, although definite cysts were not seen.

CONGENITAL LOBAR EMPHYSEMA

Thus far, cases illustrating three types of pulmonary cysts have been reported. It seems worth while to mention a condition reported in the literature as a definite entity that clinically and roentgenologically simulates closely pulmonary cyst. The treatment is so different that a specific diagnosis is imperative.

CASE 9.—H. D., a 5 months old white boy, developed normally for the first 3 months of life but for the last 2 months had not eaten well and had failed to gain weight. Just prior to hospital admission difficult respiration was noted, and infection of the upper part of the respiratory tract occurred. Roentgenographic examination (fig. 8a) showed a pronounced decrease in density in the right lung field and displacement of mediastinal structures to the left; on a lateral roentgenogram there was a herniation through the anterior mediastinum from the right to the left. Cyanosis occurred when the infant cried. A diagnosis of expansile cyst was made, and surgery was advised. After several days of

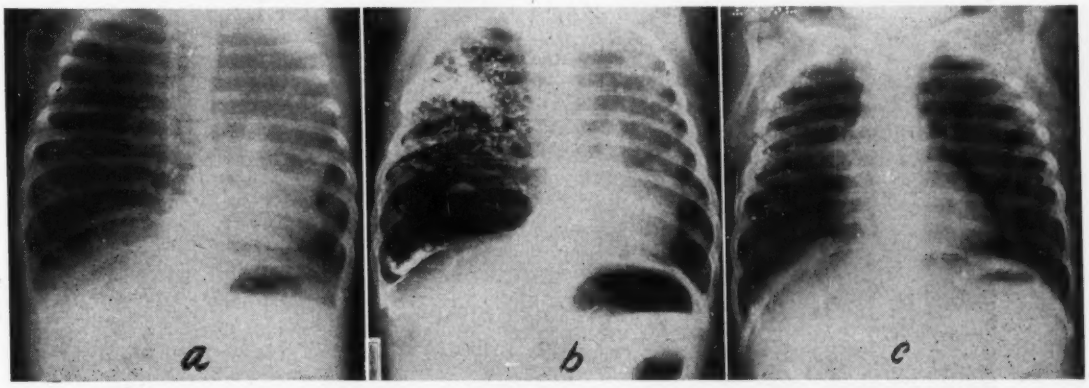

FIG. 8. Case 9. a. Congenital lobar emphysema. The middle lobe of the right lung occupies the entire right hemithorax and herniates to the left side.

b. Two days later, attempts to decompress the "cyst" and to outline the cavity with oil prove the diagnosis of lobar emphysema.

c. Twelve months after removal of the emphysematous middle lobe of the right lung, minimal pleural scarring is evident.

antibiotic therapy and oxygen no improvement was noted in the child's condition. An attempt was made to decompress the cyst; however, the needle did not enter a true cystic cavity. Iodized oil was injected, and roentgen examination (fig. 8b) revealed that some of the opaque medium was in emphysematous lung tissue and that the pleural space was outlined.

With the information that no cystic cavity was entered by the needle, the initial roentgenograms were restudied, and vascular markings could be seen over the area previously thought to be a cystic sac. This review resulted in a change of diagnosis to congenital lobar emphysema. The right side of the chest was incised, and the middle lobe was noted to occupy almost the entire right hemithorax and to herniate through the anterior mediastinum to touch the left lateral chest wall. The upper and lower lobes on the right side were completely collapsed. The middle lobe was removed, and the remaining lobes were expanded. The lower lobe expanded rapidly but the upper lobe enlarged with difficulty, giving the impression that it had never been previously aerated. Microscopic examination of the surgical specimen revealed diffuse emphysema throughout the middle lobe with some ruptured alveoli.

The postoperative course was normal. Immediately after surgery the respiration was observed to improve, and according to the parents the rate was slower and more nearly normal than it had ever been before. The child was dismissed from the hospital on the eighth postoperative day. In the year after the operation he developed normally in every respect. There have been no respiratory infections of any significance, and chest roentgenograms (fig. 8c) made almost a year after the lobectomy showed only minimal pleural reaction and a small amount of iodized oil remaining in the pleural space. The final diagnosis in this case was congenital lobar emphysema involving the middle lobe of the right lung.

Cases 5, 6, 7, and 8 illustrate cysts occurring in infants and young children, and case 9 is of congenital lobar emphysema, which must be differentiated from air cysts for proper treatment. Both of these conditions occur during the period of development of the lungs. It may be that some insult during this period of rapid development is responsible for the occurrence of cysts of the type described as well as of lobar emphysema. These cases pose problems of diagnosis and of management, and patients must be carefully watched and vigorously treated if success is to be obtained. However, they are few in number.

EMPHYSEMATOUS BLEBS

The next 3 cases illustrate the type of cystic sacs that may occur in an older age group, usually associated with emphysema, and may be considered as degenerative changes, in contrast to those occurring during the age of lung development. The higher incidence of emphysematous changes in the lung that is being observed has been attributed to increased longevity, exposure to irritants, and respiratory infections. The patient with slow but unmistakable increase in dyspnea and decreasing tolerance to exercise progressing to the point of complete disability presents a tremendous problem, both from the medical and economic standpoint. Although only a small percentage of this large group of unfortunate patients have localized cystic areas amenable to active treatment, it is extremely important to recognize and treat them as indicated since rehabilitation can be complete.

CASE 10.—G. F. was a 67 year old white man who had noted increasing dyspnea upon exertion for several years. There was present each morning a cough productive of thick mucoid sputum. Wheezing had been present for two years, and there was some orthopnea. A chest radiograph made on hospital admission (fig. 9a) showed replacement of essentially all normal lung tissue in the left hemithorax by one or more air sacs. Bronchoscopy and bronchograms revealed that the cysts were chiefly in the upper lobe and that some of the lower lobe had collapsed against the mediastinum.

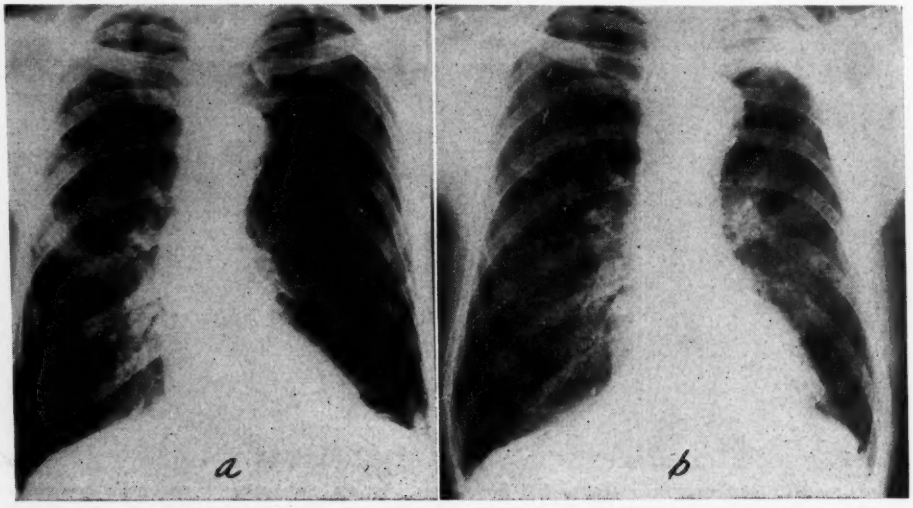

FIG. 9. Case 10. a. A cyst of the upper lobe of the left lung occupies almost all of the left hemithorax in this roentgenogram. b. Eight months after treatment by suction drainage and Lipiodol injection the left lung shows little emphysema and fibrosis.

Surgical removal of the upper lobe was thought advisable, but the cardiologist advised against major surgery. Accordingly, the cyst was decompressed by suction drainage through an intercostal catheter. With suction there was gradual reduction in the size of the cyst but little expansion of the remaining lung. In an effort to promote scarring and obliteration of the bronchial communications of the cyst, iodized oil was introduced into the cavity and suction was discontinued. Shortly thereafter, infection occurred within the cyst with outpouring of fluid, fever, and respiratory distress. Drainage of the pleural space and of the cyst was reinstituted, and a large quantity of fluid removed.

Improvement gradually occurred, and the patient was dismissed after three weeks of hospital treatment. Clinically, he was much improved; he returned to work and was able to live a normal life for a man of his age. A roentgenogram (fig. 9b) made eight months after suction drainage showed some residual Lipiodol in the pleural space. The left lung was well aerated and showed surprisingly little emphysema and fibrosis. After two years of normal life, the patient died of a strangulated hernia.

CASE 11.—E. O. H., a 59 year old white man, was admitted to the hospital with the history of dyspnea and orthopnea for two years. He had a cough present in the mornings, thick mucoid sputum, and some loss in weight. The cough had been present for many years, and dyspnea had been progressive.

A roentgenogram on admission (fig. 10a) revealed several large cysts in both upper lung fields. The diaphragm moved fairly well, suggesting that some functioning pulmonary tissue was present and that improvement might be expected from removal of the large cystic areas in the upper lobes. The right side was operated upon; two large cysts and numerous smaller cysts were observed. The walls of the large cysts were resected; the small cysts were punctured and plicated. The postoperative course was complicated by rather slow expansion of the lung. Roentgenograms at three months (fig. 10b) showed the right lung to be noticeably improved; the cysts on the left remained the same size, and there was a small amount of fluid present. The original plan was to operate upon the left side at a later date, but the patient suffered a coronary occlusion. Respiratory symptoms have decreased since operation.

CASE 12.—R. L. N., a 45 year old white man, was admitted to the hospital with a history of cough and weakness of from three to four months' duration, low grade fever, and production of large quantities of sputum in the morning. Nine years prior to admission of the patient respiratory symptoms began after a severe attack of measles. One year later there was severe chest pain with cough and fever, and since that time wheezing and production of sputum. Hemoptysis had occurred frequently. Admission roentgenograms revealed extensive changes in the right side with overdistention of the lower lobe, irregularities about the diaphragm, and cystic cavities in the upper lung field. The left lung appeared relatively normal. At exploratory thoracotomy, considerable pleural adhesions were present with thickening of the visceral pleura. The entire right lung was honeycombed with cysts. Several cysts in the upper lobe were deroofed, and the entire lung was decorticated.

The postoperative course was entirely satisfactory, and the patient improved considerably. A roentgenogram made about two years after operation showed better position of the right diaphragm and a more nearly normal appearance of the entire right lung field. The patient has returned to his vocation as a farmer in the summer and a commercial fisherman in the winter.

DISCUSSION

In many pulmonary cysts it is impossible to determine by clinical or pathologic examination whether the cyst was congenital or acquired and whether it originated in a bronchus or in the alveolar structures of the lung. More frequent pathologic examinations of these lesions, brought about principally by advancements in thoracic surgery, may throw much additional light upon their etiology as well as upon the best methods of treatment. The recognition of pulmonary cysts is dependent upon the frequent and judicious use of roentgen examinations in appraising

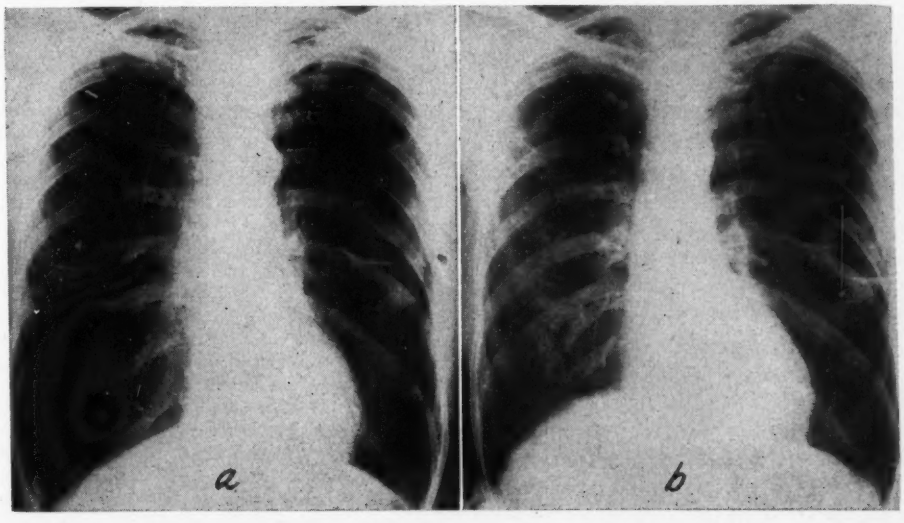

FIG. 10. Case 11. a. Bilateral emphysematous cysts occupy the upper lung fields in this admission roentgenogram. b. Three months after surgical treatment of the right side, that lung is noticeably improved. The left lung is about the same.

the efficacy of therapeutic measures in pulmonary conditions, particularly those of children.

Cysts that have bronchial communications become infected, and eradication of this infection is not complete until the cyst is removed by surgery or spontaneous obliteration. It seems probable that all individual cysts originate in a single lobule of the lung. Adjacent healthy lung tissue suffers varying degrees of damage, depending upon the duration of the cyst, its size, and the intensity of the infection. Therefore, the conservative treatment of a cyst that compresses adjacent normal lung tissue should not be continued long enough to permit irreparable damage which in the end will result in the sacrifice of a greater amount of lung tissue or in permanent crippling of the patient. The developing lung of young infants and children is more prone to form cysts, but in these patients the efficacy of conservative measures by the optimistic pediatrician and persevering thoracic surgeon has resulted in a clearing of a large percentage of cysts without sacrificing lung tissue. It is in this group that rapidly expanding cysts may occur, resulting in asphyxia and death if surgical intervention is not prompt. Such an instance was reported by Anspach and Wolman[1] in 1933, by one of the authors[6] in 1937, and by many others in more recent literature.

Congenital lobar emphysema, which has been recognized more recently as a pathologic entity, presents a clinical course and radiologic signs that closely simulate expansile air cysts. A differentiation between the conditions is important because the best treatment of congenital lobar emphysema seems to be surgical removal of the lobe as soon as the diagnosis is made.

The ingestion or aspiration of volatile oils may be followed by serious pulmonary disease. The condition warrants close observation and clinical management until the lungs have returned to normal as determined by roentgenologic as well as clinical examination.

A group of pulmonary diseases which might be classified as degenerative disease is characterized by overdistention of lung tissue and a progressive loss of respiratory reserve. This in future years may become by far the most important medical and economic problem of all pulmonary diseases.

As the radiologists, internists, and general practitioners begin to devote more attention to the prevention and treatment of pulmonary cysts, one can expect greater things to come from the surgical removal of the larger cystic sacs as well as from the newer and more effective methods of coping with the smaller cysts before all of the salvageable lung tissue has been affected.

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ABSTRACT OF DISCUSSION

DR. MARTIN SCHNEIDER, Galveston: As a study of lung development has progressed, it has become apparent that classifications of pulmonary cysts have varied because of the difficulty of differentiating prenatal from postnatal lung changes, and these in turn from the effects of infection or edema. It is known that pulmonary development begins with the 3 mm. embryo and continues by linear growth, by increase in caliber of the bronchial tree, and by a series of divisions until a person is about the age of 14 years. Obviously, then, one should speak of developmental, rather than congenital, abnormalities to emphasize the fact of continuous lung growth in the fetus and child.

Dr. King and Dr. Cole's paper is a timely reminder of the essential clinical approach to the management of the patient with pulmonary cystic disease at any age. One gathers from this presentation that serial radiologic examinations play one of the major roles in the decision to treat such patients by medical means or by surgery. There exist emergency situations in which either developmental or postinfectious cysts may become tension cysts threatening the life of the patient by suffocation. There are other situations in which repeated infections of the lung and pleura, or repeated hemoptysis, also threaten the patient's life by the disability imposed on him from their cumulative effect. The question of therapy is resolved by consideration of the patient's general condition, the effect of cystic disease on the lung function, and the status of the disease as a progressive or a static condition. With an infected cyst which does not respond to medical measures, it is surely more important to adopt a correct surgical procedure and treat the lesion by puncture and Monaldi drainage or, preferably, by resection than to question whether a developmental cyst with secondary infection or an infection with secondary cyst is present.

I do not mean to imply, however, that careful study of the lung ought not to be performed first. Bronchography or laminography may reveal the presence of more cysts or of cystic bronchiectasis and critically influence the choice of therapy. Radiologic demonstration of an anomalous systemic blood supply to a so-called "sequestrated" lobe or segment is indispensable to the surgeon in planning his procedure. Fluoroscopy may yield information to help differentiate large cysts or pneumatoceles from pneumothorax. I acknowledge with thanks my indebtedness to the thoracic surgeons for an increasing appreciation of the ultimate significance of monocystic and multicystic pulmonary lesions, even in the asymptomatic stages.

I suppose no discussion is really complete without expressing some differences of opinion, even with as excellent a presentation as this one. Thus, I question the classification

of case 9 as congenital lobar emphysema; to me, the history of preceding respiratory infection and the pathologist's report of ruptured alveoli throughout an emphysematous right middle lobe indicates a simple postbronchitic obstructive emphysema in well developed lung tissue, not uncommon when edema and/or inflammation produces a check-valve in a bronchus. In case 12, the sequence of events suggests a postpneumonic saccular bronchiectasis, though preexisting and asymptomatic developmental bronchiectasis may have existed; however, I have difficulty reconciling it with the author's group of degenerative emphysematous cysts.

At John Sealy Hospital, we found no pulmonary cysts after kerosene pneumonitis in 36 cases; I wonder if case 8 is an exceptional or a common type in Dr. King's experience.

My preference for a classification would be one grouping of developmental cysts, with or without secondary infection or obstruction (partial or complete), and a second grouping of acquired cysts secondary to infection or to obstruction of the bronchial tree from edema, inflammation, new growth, or foreign body.

DR. KING, closing: Our experience with cases of pneumonitis after the ingestion or aspiration of volatile oils has been similar to that of Dr. Schneider, that is, practically all infections cleared completely with no residual disease. Case 8 is the only one in which cystic air sacs and chronic infection developed.

Differential Diagnosis of Mild Painless Jaundice

JAMES C. CAIN, M.D., Rochester, Minnesota

PATIENTS who have mild, painless jaundice are commonly encountered and they always place the physician in a diagnostic predicament. Every type of jaundice, at some time in its course, may be mild and painless, but the true character of the jaundice usually becomes evident in the course of time.

The first requisite, therefore, is to cut the subject down to size. For the present purpose then, "pain" may be considered to mean spontaneous pain, not pain elicited by the examiner's hand, and "painlessness" to mean absence of spontaneous pain. To judge the degree of jaundice by inspection is not reliable. Apparent jaundice may not be jaundice at all, even in the presence of hepatic disease. The darkening of the skin may be attributable to derangement of melanin metabolism. Therefore, in estimating the degree of jaundice it is best to rely on determination of the value for serum bilirubin. Mild, painless jaundice will be said to exist if the value for total serum bilirubin lies between 1.5 and approximately 14 mg. per 100 cc. For this discussion only those types of jaundice which are persistently mild and painless will be considered. They are as follows:

Obstructive jaundice
- Stone in the common bile duct
- Carcinoma of the ampulla of Vater
- Stricture of the common bile duct
- Biliary cirrhosis

Hepatocellular jaundice
- Hepatitis
  - Infectious hepatitis
  - Serum hepatitis
- Portal cirrhosis
- Constitutional hepatic dysfunction

Hemolytic jaundice

It is understood that this list is oversimplified. These few examples, however, allow the main problems to be presented. More detailed consideration in a short time and space is impracticable. The magnitude of the problem which faces the diagnostician is suggested in tables 1 and 2. Detailed discussion of the classification, etiology and pathologic physiology involved in jaundice are of great importance in understanding the problem but do not seem to be pertinent to brief discussion of the differential diagnosis of mild, painless jaundice.

When confronted with a patient who has mild, painless jaundice, the physician's major problem is to determine whether the patient requires an immediate operation or whether he should be treated nonsurgically. This is of more than academic interest, for the life of the patient who has hepatocellular disease will be jeopardized by a surgical procedure. On the other hand, the condition of the patient who has obstructive jaundice, if treated conservatively for a long time, will become much worse and perhaps he will miss the chance for complete cure by surgical measures.

HISTORY

An accurate and detailed history must be obtained, and in about three-fourths of the cases of mild, painless jaundice this will lead to an accurate impression of what condition is present. Has the patient been in contact with any hepatotoxic agent, such as alcohol or chloroform, which could be responsible for cirrhosis? Has the patient associated with other jaundiced patients, whose condition might be attributable to infectious jaundice? Has the patient received any subcutaneous injections that might contain blood prod-

From the Division of Medicine, Mayo Clinic.

Read before the Section on General Practice, Texas Medical Association, Annual Session, Houston, April 28, 1953.

ucts? The virus of infectious hepatitis may be transmitted by as little as .01 cc. of infected serum. Thus far, the physician has been asking questions, affirmative answers to which give him the impression that some form of hepatocellular disease may be present.

Can a chronologic history of attacks of gallbladder disease be obtained? Has the patient undergone operative procedures involving the gallbladder or common bile duct? If so, was difficulty encountered at operation or was the postoperative course complicated? Does the patient tell of attacks of fever, chills, jaundice, or itching, related to passage of dark urine? These symptoms suggest an obstructive type of jaundice, attributable to stone in the common bile duct, stricture of the common bile duct, or carcinoma of the ampulla of Vater.

A family history of blood dyscrasia, or symptoms suggestive of congenital hemolytic icterus, suggest that the patient's difficulty is hemolytic jaundice.

PHYSICAL EXAMINATION

A carefully performed physical examination will be of considerable aid. Estimation, by inspection, of the degree of jaundice is often inaccurate, as has been said, but the skin, scleras, and mucous membranes should be inspected nevertheless. The presence, moreover, of angiomatous spiders, palmar erythema, abdominal varices, fine thin hair, gynecomastia, testicular atrophy, and fetor hepaticus suggests the diagnosis of hepatocellular jaundice. Huge size of a liver often is attributable to metastatic carcinoma, hepatoma, or perhaps primary or secondary biliary cirrhosis. An enlarged spleen suggests that the jaundice is of hepatocellular type and helps to exclude jaundice attributable to carcinoma or to early obstruction of the common duct. A palpable gallbladder often indicates complete obstruction of the bile duct by carcinoma, either carcinoma at the head of the pancreas or carcinoma of the common bile duct.

LABORATORY DETERMINATIONS

The history and physical examination give the clinician strong grounds for diagnosis, but a clinical impression should be reinforced by confirmatory laboratory examinations.

The concentration of serum bilirubin should be determined. This will give accurate measurement of the degree of jaundice and, by use of the van den Bergh test, the exact amount of direct and indirect reacting bilirubin will be learned. If less than .2 mg. of the bilirubin per 100 cc. reacts directly, and the remainder indirectly, the suggestion is that the jaundice is attributable either to a hemolytic process or to constitutional hepatic dysfunction. The quantity of uro-

TABLE 1.—*Classification of Obstructive and Hepatocellular Jaundice.*

- EXTRAHEPATIC OBSTRUCTIVE JAUNDICE
  - Benign obstruction
    - Stone in common bile duct
    - Stricture of common bile duct
    - Pancreatitis
  - Carcinomatous obstruction
    - In head of pancreas
    - In ampulla of Vater
    - In bile ducts
      - Below junction of hepatic bile duct and cystic duct
      - Above junction of hepatic bile duct and cystic duct
- HEPATOCELLULAR JAUNDICE
  - Hepatitis
    - Viral forms
      - Infectious hepatitis
      - Serum hepatitis
      - Yellow fever
      - Dengue
      - Infectious mononucleosis
      - Unidentified
    - Spirochetal forms
      - Syphilis
      - Weil's disease
    - Bacterial forms
      - Septicemia
      - Brucellosis
      - Typhoid
    - Parasitic forms
      - Ameba
      - Echinococcus
      - Liver fluke
    - Toxic forms
      - Systemic disease
        - Exophthalmic goiter
        - Toxemia of pregnancy
      - Drugs
        - Arsenic
        - Cinchophen
        - Chloroform
        - Chiniofon
        - Sulfonamides
      - Industrial poisons
        - Phosphorus
        - Picric acid
        - Arsenic
        - Dinitrophenol
        - Carbon tetrachloride
        - Secondary to obstruction
  - Cirrhosis
    - Primarily fatty in pathogenesis
      - Dietary deficiency (kwashiorkor)
      - Chronic alcoholism and dietary deficiency, Laennec type
      - Toxic fatty liver (arsenic, carbon tetrachloride, phosphorus, certain systemic diseases)
      - Diabetic fatty liver
    - Primarily nonfatty in pathogenesis
      - Viral or idiopathic
        - Postnecrotic (toxic changes or coarsely nodular liver, healed acute atrophy)
        - Diffuse portal (chronic hepatitis with fibrosis, mainly portal)
          - With hepatocellular impairment
          - Cholangiolitic (primary biliary cirrhosis, Hanot's type)
        - Transitional and mixed
      - Parasitic—schistosomiasis
      - Syphilitic—probably only hepar lobatum
      - Brucellar (?)
      - Obstructive biliary (cholostatic and cholangitic)
      - Carcinomatous, metastatic in liver
      - Metabolic error
        - Hemochromatosis ("pigmentary" cirrhosis)
        - Wilson's disease, Fanconi's syndrome } amino-aciduria
        - Porphyria hepatica
      - Cardiac (central necrosis and fibrosis resulting from long-standing chronic passive congestion)
  - Constitutional hepatic dysfunction. (Note: This condition sometimes is included under hepatocellular jaundice, as in this table; sometimes it is considered as a condition distinct from hepatocellular jaundice, as it is in the text of this paper. In any case it is a dysfunction, not a disease.)

bilinogen in a 72 hour specimen of stool (reported on the basis of a 24 hour fraction) will accurately separate these two conditions. In the 24 hour stool of a patient who has hemolytic icterus more than 300 mg. of urobilinogen will be found. In the stool of a patient with constitutional hepatic dysfunction the quantity of urobilinogen will be normal (40 to 280 mg. per 24 hours). Constitutional hepatic dysfunction is not actually a disease of the liver and results of all laboratory examinations, except an elevated value for indirect reacting bilirubin, are normal. It is important that this condition be recognized for what it is—a dysfunction. It need not be considered again until the reports of illustrative cases, and the reader who wishes to look ahead will find this condition illustrated in the report of case 1.

TABLE 2.—*Classification of Hemolytic Disorders.*

EXTRACORPUSCULAR CAUSES
Infectious agents
Malaria*
Bartonella (Oroya fever)*
Septicemia: Clostridium welchii, Streptococcus pyogenes, Streptococcus mitis, Escherichia coli, Pneumococci, Staphylococci
Chemical agents
Phenylhydrazine
Allyl-propyl-disulfide
Saponin
Methyl chloride
Paraphenylenediamine
Colloidal silver
Toluene
Benzene
Arseniuretted hydrogen
Trinitrotoluene
Dinitrobenzene
Naphthalene
Aniline
Phenol compounds
Acetanilid
Phenacetin
Sulfonamides
Quinine
Pamaquine
Lead
Physical agents (heat—severe thermal burns)
Vegetable and animal poisons
Vegetable poisons
Fava bean (Vicia faba)
Castor bean (ricin)
"Baghdad anemia"
Animal poisons
Snake venoms
Endogenous agents
Hemolytic anemias due to immune body reactions
Isoagglutinins
Anti-A, anti-B (transfusion reactions)
Anti-Rh, anti-Hr, M, and other forms (hemolytic disease of the newborn, "intragroup" transfusion reactions)
Cold hemolysins (paroxysmal cold hemoglobinuria)
Cold, warm, and blocking antibodies
Idiopathic "acquired" hemolytic anemia without demonstrable hemolysins or agglutinins
Secondary, symptomatic, or "acquired" hemolytic anemias
Hodgkin's disease
Leukemia
Lymphosarcoma
Metastatic carcinomatosis
Dermoid cysts
Liver disease, and so forth
INTRACORPUSCULAR DEFECTS
Familial or congenital hemolytic jaundice (hereditary spherocytosis)
Sickle cell anemia
Paroxysmal nocturnal hemoglobinuria
"Mediterranean" anemia
Hereditary nonspherocytic hemolytic anemia

From Wintrobe, M. M.: Clinical Hematology, ed. 3, Philadelphia, Lea & Febiger, 1951, p. 565.

*In these instances the causative agents, while originally extrinsic, actually produce erythrocyte destruction through their presence in the red corpuscles; the osmotic and mechanical fragilities of parasitized corpuscles have been shown to be increased.

The causes of hemolytic icterus are many (table 2). These diseases are classified primarily as hematologic problems and these, again, will not be discussed further.

If the serum contains more than .2 mg. per 100 cc. of direct reacting bilirubin, a hepatocellular or an obstructive type of jaundice is present. The proportions of direct and indirect reacting bilirubin are of little or no value in distinguishing between these two large groups of jaundiced patients. If the value for total serum bilirubin is more than 10 mg. per 100 cc., complete obstruction of the common bile duct is possible.

If bile is not obtained on repeated duodenal drainage, complete obstruction of the common bile duct is likely. This should be confirmed by determining the amount of urobilinogen in the stool. A 72 hour specimen of stool noted to contain less than 10 mg. of urobilinogen per 24 hour fraction further verifies the diagnosis of complete obstruction of the common bile duct. Such obstruction is likely to be attributable to carcinoma of the head of the pancreas, carcinoma of the common bile duct, or perhaps complete surgical stricture of the common bile duct. Patients whose common bile ducts are completely obstructed have more than mild jaundice and can be eliminated from this discussion.

Severe hepatocellular jaundice may markedly suppress the secretion of bile for from seven to ten days, but repeated duodenal drainage will disclose that bile is passing through the duct. If bile is obtained by duodenal drainage, the indication is only that complete obstruction of the common bile duct is absent. If the bile contains blood, this is suggestive evidence of carcinoma of the ampulla of Vater. By duodenal drainage, Miller and his associates obtained blood that was evident on gross inspection from approximately 38 per cent of patients with tumor of the ampulla of Vater.

Thus, constitutional hepatic dysfunction, the hemolytic types of jaundice, and complete obstruction of the common bile duct have been identified or excluded. The patient with incomplete obstructive jaundice and the patient with hepatocellular disease still need to be distinguished one from the other.

If the value for serum alkaline phosphatase is greater than 12 to 14 Bodansky units, the presence of an obstructive type of jaundice is suggested. (The King-Armstrong alkaline phosphatase unit is approximately three times that of the Bodansky unit.) Values for alkaline phosphatase of less than 12 Bodansky

units are not of much aid, for they may be found in the presence either of obstructive or hepatocellular jaundice.

Values for serum cholesterol and for cholesterol esters may be more than 300 mg. per 100 cc. in the presence of obstructive jaundice. Concentrations of cholesterol esters less than 30 per cent of the total value for serum cholesterol suggest serious hepatocellular disease. Normal values for serum cholesterol and cholesterol esters are of no differential significance because they are found in the presence both of obstructive jaundice and hepatocellular jaundice.

The prothrombin time may be prolonged in the presence either of obstructive jaundice or of hepatocellular jaundice. If the prolonged prothrombin time returns to normal within twenty-four hours after ingestion of 4 mg. of water-soluble vitamin K, presence of an obstructive type of jaundice is strongly suggested. If the prolonged prothrombin time returns to normal slowly or fails to become normal, the presence of hepatocellular disease is suggested.

Thus, an elevated value for serum alkaline phosphatase, an elevated value for serum cholesterol, and a prolonged prothrombin time that responds to oral administration of vitamin K are positive tests indicating that the jaundice is probably of obstructive origin.

Many laboratory determinations help confirm the presence of hepatocellular jaundice. The flocculation tests (so-called liver function tests) frequently give positive results in the presence of hepatocellular disease. Many of these tests have been described but at Rochester we have found three most useful.

The result of the cephalin-cholesterol flocculation test is usually more than 2 plus when the patient has hepatocellular disease. Unfortunately this test may also be positive in the presence of obstructive jaundice. It will be positive if an insufficient amount of serum albumin is present to inhibit the flocculation of the cephalin-cholesterol. It does not give an index of the severity of hepatic damage and actually is not a true test of hepatic function.

The thymol turbidity test apparently depends on derangement of the serum lipids, the lipoprotein complexes, and the gamma globulin fractions of the serum. Its result usually is within normal limits (0 to 4 units) if patients have an obstructive type of jaundice. It may be positive in association with many diseases that cause reticuloendothelial irritation, and in the presence of hepatocellular disease, the result is often above normal. When the result of this test is greater than 4 units although the other findings suggest obstructive jaundice, associated hepatocellular disease is very likely.

The Kunkel test for gamma globulin may be employed. Values of more than 16 units suggest the presence of hepatocellular disease, but such values may be found in association with any disease that causes elevation of the values for gamma globulins.

A decrease in the value for total serum protein suggests the presence of hepatocellular disease, and the ratio of albumin to globulin may be reversed. In normal human serum the concentration of protein usually is about 7.5 Gm. per 100 cc., and normally 60 to 65 per cent of the protein is albumin.

Mild anemia often is present in association with hepatocellular disease accompanied by mild, painless jaundice. A blood smear frequently will show a tendency toward macrocytosis.

These various determinations help to group patients into the group of incomplete obstructive jaundice and hepatocellular disease.

Unfortunately, even when the measures mentioned are employed, the condition of some patients with mild, painless jaundice cannot clearly be diagnosed, either as obstructive jaundice or as hepatocellular jaundice. It was hoped that biopsy of the liver would be a safe, practical, and accurate method of identifying these conditions. Biopsy has been a great aid in following the course of hepatic disease and it has added much to fundamental understanding in this field, but it is not entirely safe, and accurate diagnosis cannot always be made from stained sections. Biopsy of the liver has been a positive aid in diagnosing hemochromatosis, amyloidosis, and fatty liver.[2] It also often discloses the cause of mild, painless jaundice when this condition is attributable to malignancy.

NARROWING OF DIAGNOSIS

In the foregoing discussion, for the most part, I began with the evidence and progressed to the diagnosis. Now, I shall reverse the process. The reader will notice a little repetition, but I hope it will be considered purposeful, as I believe it to be.

When the physician has determined that a patient with mild, painless jaundice has neither hemolytic jaundice nor constitutional hepatic dysfunction, but that he has either obstructive jaundice or hepatocellular jaundice, the physician has accomplished a great deal. He has practically settled the question of whether an operation is needed or whether the patient should be treated nonsurgically. However, if he can determine, further, what subdivision of obstructive or hepatocellular jaundice the disease of his patient represents, treatment can be even more intelligent, whether it be surgical or nonsurgical.

Types of Obstructive Jaundice.—The diagnosis of the type of obstructive jaundice usually can be made by reconsideration of the history and of the results of physical examination and laboratory studies. A patient with mild, painless jaundice attributable to a

stone in the common bile duct frequently will give a history of previous attacks of gallbladder disease. Only about from 10 to 20 per cent of these patients will have no pain,[3] but it is this group which is of particular concern here. The obstruction is incomplete, and there often will be a history of bouts of cholangitis associated with nausea, fever, chills, jaundice, dark urine, and itching. If the gallbladder has not been removed, the fact that it contains stones often can be determined by roentgenographic examination. At operation, a stone may have been pushed into the common bile duct. If there is a T tube in the common bile duct, a cholangiogram will demonstrate the stone.

TABLE 3.—*Significant Laboratory Determinations in Case 1.*

Determination	Inference
Sedimentation rate normal Urine free of bile No direct reacting serum bilirubin Prothrombin time normal	General health unimpaired No obstruction or hepatocellular disease
Plasma cholesterol and cholesterol esters normal Cholecystogram: no stones, normal function	No obstruction
Serum protein normal Albumin-globulin ratio normal Bromsulfalein test: no retention Thymol turbidity test normal Cephalin-cholesterol test normal Cholesterol esters not low (normal; see above) Blood smear: no macrocytosis	No hepatocellular disease
Blood smear: no spherocytosis Erythrocytic fragility normal	No hemolytic jaundice
Urine free of bile Indirect reacting bilirubin high (2.5 and 1.8 mg. per 100 cc.)	Hemolytic jaundice or constitutional hepatic dysfunction present
All determinations (see above)	Everything excluded except constitutional hepatic dysfunction

The only practical treatment is exploration of the common bile duct and removal of the stone. Case 2 is an example of jaundice from stone in the common bile duct.

The patient with carcinoma of the ampulla of Vater may exhibit symptoms exactly like those of a patient whose common bile duct contains a stone. The jaundice usually is fluctuating and there will be episodes of fever and chills. The patient is likely to be from 40 to 60 years of age and the course of his illness may be distinctly retrogressive. He may be weaker, may lose more weight, and may complain more of anorexia than usually is characteristic of a patient with a stone in the common bile duct. As has been said, about 38 per cent of patients who have a carcinoma at the ampulla have grossly visible blood in material obtained on duodenal drainage.

The patient with a stricture of the common bile duct nearly always has undergone operation on the gallbladder or some other portion of the biliary tract. Usually, as has been pointed out, a history of difficult operation and the complication of postoperative drainage of bile can be obtained. The symptoms begin about the time that healing and scarring would be expected, but major symptoms may be delayed for months or perhaps even years. The obstruction is rarely complete, and the value for serum bilirubin fluctuates. Fever and chills owing to cholangitis are frequent. Itching often presents a severe problem. Case 3 is a fairly typical example of stricture of the common bile duct after biliary surgery.

Rare cases of biliary cirrhosis are encountered which, in the early stage, closely resemble cases of incomplete extrahepatic obstruction. Results of laboratory determinations may fail to give evidence of hepatocellular damage and may be consistent with the diagnosis of obstructive jaundice. Patients with such symptoms ordinarily deserve an exploratory operation to discover if the disease is primary, or if it is secondary to some extrahepatic obstruction. At exploration, however, if the condition is biliary cirrhosis, the gallbladder and common bile duct are normal. If obstruction is present, it seems diffuse and located in the smaller bile ducts within the liver itself. Case 4 is a typical example of biliary cirrhosis.

Types of Hepatocellular Jaundice.—Mild, painless jaundice may be attributable to hepatocellular disease. In table 1 are listed the many factors that can cause or accompany hepatocellular jaundice. Most of the patients who exemplify this condition have either viral hepatitis or portal cirrhosis. The history is the best aid in determining the exact nature of hepatocellular jaundice.

TABLE 4.—*Significant Laboratory Determinations in Case 2.*

Determination	Inference
Urine contained bile Direct reacting serum bilirubin (10.8 mg. per 100 cc.)	No hemolytic jaundice
Prothrombin time normal Cephalin-cholesterol flocculation test normal	No hepatocellular disease
Total serum bilirubin low (11.8 mg. per 100 cc.) Bile obtained on duodenal drainage	No complete obstruction
No blood obtained on duodenal drainage	No carcinoma of ampulla (probably)
Urine contained bile (see also above) Roentgenogram of gallbladder region: evidence of stone near common duct	Incomplete obstructive jaundice present
All determinations (see above)	Everything excluded except incomplete obstructive jaundice

Hepatitis may be divided into two large groups: homologous serum hepatitis and infectious hepatitis. The incubation period for homologous serum hepatitis, is from three to six months and that of infectious hepatitis, three to six weeks. In both, laboratory findings are about the same. About 15 to 20 per cent of patients who have received pooled plasma get homologous serum hepatitis. Irradiation of the plasma and

TABLE 5.—*Significant Laboratory Determinations in Case 3.*

Determination	Inference
Direct reacting serum bilirubin present (.7 mg. per 100 cc.)	No hemolytic jaundice
Total serum protein normal Serum albumin-globulin ratio normal Result of thymol turbidity test negative Prothrombin time normal	No hepatocellular disease
Total serum bilirubin low (1.37 mg. per 100 cc.)	No complete obstruction of common duct
Bodansky alkaline phosphatase high (19.6 units)	Incomplete obstructive jaundice present
All determinations (see above)	Everything excluded except incomplete obstructive jaundice

other methods for sterilizing it have not proved successful in preventing this infection. As has been said, .01 cc. of contaminated plasma may be enough to transmit the virus. Homologous serum hepatitis is transmitted only by mechanical placement of the virus under the skin. Infectious hepatitis is transmitted chiefly by ingestion of food or drink contaminated with fecal material that contains the virus; occasionally, it is transmitted by hypodermic injection. Case 5 is an example of infectious hepatitis.

The patient with mild painless jaundice attributable to portal cirrhosis does not usually present a great diagnostic problem. There is often a history of excessive ingestion of alcohol associated with a nutritional deficiency. Nevertheless, these have not been proved to be of etiologic significance. As has been said in part, patients may present angiomatous spiders, fetor hepaticus, palmar erythema, gynecomastia, fine thin hair, testicular atrophy, a palpable spleen, a palpable liver, and ascites. The jaundice, which appears late, usually is a bad prognostic sign. The so-called liver function tests frequently indicate serious hepatocellular disease. Case 6 is a fairly typical example of portal cirrhosis.

CASE REPORTS

CASE 1.—A white male college student, 19 years of age, reported for examination in August, 1951, because of mild "dizziness after walking" and recurrent jaundice. His health had been excellent prior to November, 1949, when he had had an illness characterized by anorexia, slight nausea, and dark urine. His physician had felt an enlarged liver and the youth had been hospitalized for ten days. For the next fifteen months his health had been exceptionally good. He was a weight lifter and prided himself upon his physique. In February, 1951, he had had another episode of illness consisting of mild malaise, vague dizziness, and passage of three or four loose, brown stools a day. His appetite had remained excellent. There had been a question of whether he had fever of low grade. His urine had been dark, and mild but definite jaundice had appeared. He had been hospitalized for three and one-half weeks, and, he said, a diagnosis of "liver disease" had been made. He again had recovered completely, but his physician had warned him to take things easy. Three months later he had had a similar episode of jaundice and the value for serum bilirubin had been 2.5 mg. per 100 cc. After this episode, a chiropractor had put him to bed for three months.

At the time of examination at Mayo Clinic he looked the picture of health and complained only of vague dizziness and jaundice. He was tense and exceedingly worried about himself as was his overly protective mother. He wanted to know the status of his "liver disease" and whether he should enroll in college. Review of the family history revealed that one uncle had been subject to peculiar attacks of mild jaundice that did not seem to cause him much trouble.

The patient was 74 inches (188 cm.) tall and weighed 175 pounds (79.4 Kg.). He had lost 15 pounds (6.8 Kg.) in the past two years. The scleras were mildly icteric. The liver and spleen were not palpable and careful physical examination gave entirely negative results.

TABLE 6.—*Significant Laboratory Determinations in Case 4.*

Determination	Inference
Urine contained bile, grade 2+ Direct reacting serum bilirubin present (6.8 mg. per 100 cc.)	No hemolytic jaundice
Total serum bilirubin low (9.5 mg. per 100 cc.) Bile obtained on duodenal drainage	No complete common duct obstruction
Serum albumin-globulin ratio low (3.73:3.64 Gm. per 100 cc.) Thymol turbidity high (11 units)	Hepatocellular disease present
Bodansky alkaline phosphatase high (26.6 units) Plasma cholesterol high (467 mg. per 100 cc.) Plasma cholesterol esters normal Plasma phospholipids high (756 mg. per 100 cc.) Total plasma fatty acids high (810 mg. per 100 cc.) Total plasma lipids high (1,277 mg. per 100 cc.) Liver biopsy consistent with primary or secondary biliary cirrhosis	Incomplete obstructive jaundice present
All determinations (see above)	Results consistent with hepatocellular and obstructive jaundice suggest biliary cirrhosis

In this case, as in all others reported, laboratory work was thorough, but only significant determinations are given in table 3.

This patient was considered not to have hepatic disease; a diagnosis of constitutional hepatic dysfunction was made. He was advised not to be concerned about the mild jaundice that would occur from time to time, especially when he was nervous, tired, or emotionally upset. He was told to go to college and to live a normal life. A year later he reported that his general health was good.

Comment: The importance of this rare condition is that the patients can become invalids from hypochondriasis. Their vague complaints are likely to have been induced by their own anxiety added to that of their physician. The diagnosis is made on the basis of the patient's excellent physical condition and the fact that, except for an elevated value for indirect-reacting serum bilirubin, laboratory studies give entirely negative results.

CASE 2.—A white farmer, 72 years of age, was seen because of attacks of belching that had recurred for the past ten years. He never had had pain, fever, chills, or itching. The attacks had occurred every four to eight weeks, had lasted approximately an hour, and occasionally had awakened him from a sound sleep. Eating of fat and cabbage some-

times disagreed with him. The last episode had been of approximately six hours' duration and had been characterized by belching, distention, nausea, and vomiting, but no pain. Jaundice had been noted by the examiner but the patient had not observed it. Generally the man had felt well except that he had to urinate five or six times a night. He had lost about 10 pounds (4.5 Kg.).

The man was 64 inches (162.5 cm.) tall and weighed 131 pounds (59.4 Kg.). The blood pressure was 160 mm. of mercury systolic and 85 diastolic. The scleras were yellow. A loud systolic aortic murmur and a faint thrill were present. These were consistent with a diagnosis of aortic stenosis. The edge of the liver was just palpable. The spleen was not felt. Significant laboratory findings are recorded in table 4.

The patient was hospitalized for three or four days. The jaundice did not seem to fluctuate and the decision was to operate even though the patient's age and calcareous heart disease enhanced the risk. Operation confirmed the preoperative diagnosis of obstructive jaundice from a stone in the common bile duct. In addition, there was evidence of acute cholecystitis and cholelithiasis. The common duct was about 1 cm. in diameter and its wall was thickened. A T tube, which was sutured in place, was allowed to remain for two months. At the end of that time a choledochogram gave no evidence of obstruction to the flow of dye in the common duct. The patient was freed of his attacks of distention, belching, and nausea. He died ten years later of an unrelated condition.

TABLE 7.—*Significant Laboratory Determinations in Case 5.*

Determination	Inference
Direct reacting serum bilirubin present (7 mg. per 100 cc.)	No hemolytic jaundice
Total serum bilirubin low (8.2 mg. per 100 cc.)	No complete obstruction of the common bile duct
Bodansky alkaline phosphatase normal	No incomplete biliary obstruction
Cholesterol normal	
Thymol turbidity high (19 units)	Hepatocellular jaundice present
Cephalin-cholesterol flocculation high (4+)	
Cholesterol esters low (5.2 mg. per 100 cc.)	
Zinc sulfate gamma globulins high (26 units)	
All determinations (see above)	Laboratory evidence suggests hepatocellular disease

Comment: The decision to operate was made with dispatch. Nothing was to be gained from prolonged observation and batteries of so-called liver function tests. The patient was encouraged to eat; he was given water-soluble vitamin K orally and was observed only long enough to be sure that he could stand the operation.

CASE 3.—A white, married woman, 61 years of age, was admitted to the clinic in January, 1953, complaining of jaundice, itching, and recurring fever and chills. She had had typhoid fever at the age of 11 years. Rather typical attacks of cholecystitis had occurred in 1928 and had recurred infrequently. Operation performed for stones in the gallbladder had been difficult, and soon afterward mild jaundice had appeared and pruritus had developed. She had continued to have trouble, and in July, 1945, an abscess containing 3,000 cc. of bile-stained pus had been drained. The itching had become even more intense. In September, 1945, anastomosis of the hepatic duct and duodenum had been effected. Three days later, the itching had been completely relieved. Thereafter, the patient had gained weight and had lived a normal, active life.

In 1951, however, the symptoms which caused her to seek further advice had begun. She again had had recurrent fever and chills without pain. The chills and fever would last for a few hours and in a day or so the urine would be dark and mild pruritus would begin. These attacks of fever and chills had occurred every few months; they had increased in frequency until, for the month prior to the patient's admission at the clinic, she was having continuous severe itching, mild jaundice, and, at least two or three times a week, chills and fever. There was no abdominal pain.

She was 69.5 inches (176.5 cm.) tall and weighed 136 pounds (61.7 Kg.). She had lost approximately 20 pounds (9 Kg.) in the past year. The blood pressure was 169 mm. of mercury systolic and 92 diastolic. The scleras were mildly icteric. The right lobe of the liver was palpable 2 cm. below the costal margin; the spleen could not be felt. Scratching had caused dermatitis. Angiomatous spiders, palmar redness, and abdominal varices were absent. The clinical impression was that the patient had a recurrent stricture of the common bile duct. Results of significant laboratory determinations were consistent with this impression and are given in table 5.

The patient's general condition was good and on January 23, 1953, hepaticolithotomy and hepaticoduodenostomy were performed. A recurring stricture at the site of hepaticoduodenostomy was found, along with intrahepatic lithiasis. The liver was slightly enlarged but the spleen appeared essentially normal except that it was encased in a great many adhesions. Two Mayo-Sullivan tubes were sewed in place. It was thought that the catgut sutures would absorb in about six weeks to three months and that, by that time, the likelihood of stricture of the hepatic ducts would have passed.

Comment: It is of particular interest that this patient got along well for about six years. Probably there was some narrowing at the site of anastomosis of the

TABLE 8.—*Significant Laboratory Determinations in Case 6.*

Determination	Inference
Urine contained bile	No hemolytic jaundice
Direct reacting serum bilirubin present (7.8 mg. per 100 cc.)	
Total serum bilirubin low (9.7 mg. per 100 cc.)	No complete obstructive jaundice
Bodansky alkaline phosphatase normal	No obstructive jaundice
Plasma cholesterol normal	
Prothrombin time prolonged (26 sec.). After oral administration of water-soluble vitamin K prothrombin time remained prolonged (24 sec.)	
Total serum protein normal	No hepatocellular disease
Albumin-globulin ratio normal	
Thymol-turbidity test normal (4 units)	
Cephalin-cholesterol flocculation test normal (2+)	
Prothrombin time prolonged (26 sec.). After oral administration of water-soluble vitamin K prothrombin time remained prolonged (24 sec.)	Hepatocellular disease present
Liver biopsy: Portal cirrhosis, grade 3, with fatty changes	
All determinations (see above)	Laboratory determinations are not conclusive but the positive tests suggest hepatocellular disease

hepatic duct and the duodenum during all this time but not enough to cause more than some dilatation of the duct. This may or may not have played a role in the formation of stones in the duct. These stones, plus the stricture, were enough to result in recurrent, painless jaundice associated with fever and chills.

CASE 4.—A white housewife, 46 years of age, complained only of jaundice and mild pruritus. Her past health had been excellent. She had not been in contact with jaundiced patients nor had she had any subcutaneous injections for six months prior to the onset of jaundice. The jaundice had come on insidiously and possibly had begun eleven months prior to her admission to a hospital affiliated with the clinic. About the time when the jaundice had begun, her temperature had risen to 102 F. and she had had generalized aching. The entire family had had similar difficulty at the same time but it had lasted only a few days. The patient's appetite had remained good, and there had been no change in her general strength. Because of the mild jaundice she had called on her physician, who had found the liver to be enlarged and the flocculation (so-called liver function) tests to give abnormal results. The patient felt that the jaundice had fluctuated but never had cleared completely. The urine had been dark on occasions, but the stools had not been light-colored. The itching had been troublesome but never severe. There may have been some mild weakness and lassitude, but it never had been marked. As the months passed the patient had become concerned about herself even though she felt fairly well.

The woman was 51.5 inches (131 cm.) tall and weighed 122 pounds (55.3 Kg.). The blood pressure was 128 mm. of mercury systolic and 74 mm. diastolic. The skin was dark and the scleras definitely icteric. Excoriations of the skin were noticeable. There were no angiomatous spiders and there was no palmar redness, fetor hepaticus, or xanthomas. The abdomen was not enlarged and there were no abdominal varices; ascites was absent. The liver was palpable 2 cm. below the costal margin. The spleen could not be felt. Otherwise physical examination did not disclose anything abnormal. Results of significant laboratory studies are given in table 6.

The diagnosis of biliary cirrhosis was obvious. The cirrhosis could be secondary to obstruction from stone, but since there had been no previous surgery it likely would not be secondary to obstruction from stricture. The question of the advisability of a careful exploratory operation to discover whether a stone is present is always pertinent in these cases, as has been said. Surgery was offered this patient but it was not urged because clinically, the diagnosis of primary biliary cirrhosis seemed fairly certain. The patient elected not to have the operation. Accordingly, she was advised to follow a diet high in calories—carbohydrate and protein. It was recommended that she take vitamin supplements, including 4 mg. each day of water-soluble vitamin K. She was advised to restrict her activities but it was not believed that complete rest in bed was indicated.

About six months after this patient's dismissal, it was reported by letter that results of laboratory determinations were essentially unchanged. Her general condition remained good but it was thought that her prognosis was poor.

Comment: In cases of this kind the onset usually is insidious and the course discouraging. Patients may live for from five to ten years but generally they die in a state of hepatic decompensation similar to that of the patient who has portal cirrhosis.

CASE 5.—A white male geologist from Texas, 58 years of age, was hospitalized because of weakness of two weeks' duration and jaundice of four days' duration. Symptoms had begun when he was on board ship returning from a visit to England, France, Greece, Portugal, Spain, and Turkey. The onset had been gradual and had been characterized by generalized dull aching, malaise, anorexia, nausea, weakness, a temperature of 101 F., and several true chills. He had been treated with penicillin, Aureomycin, and Chloromycetin, and by the third day the temperature had been normal but the anorexia and nausea had persisted. A temperature of 100 F. had recurred about seven days after the original onset and had been accompanied, again, by nausea and occasionally some vomiting. Treatment with streptomycin and sulfadiazine had been instituted. Dark urine, light-colored stools, and yellow scleras were noted ten days after the original onset. There had been no rash but there had been some mild itching. The man stated that he always got hives and itching with little or no detectable provocation. The bowel habits had not been disturbed, but the stools had become light in color. He had not had diarrhea or bleeding from the bowel.

He had known for the past six years that he had a duodenal ulcer. He had drunk approximately two highballs a day for many years. He denied any contact with jaundiced patients. He had received the usual vaccinations before going overseas but he had received no other injections.

The man was well nourished. The scleras were icteric. The blood pressure was 130 mm. of mercury systolic and 90 diastolic. There were no palpable lymph nodes or masses. The liver and spleen could not be felt. Results of significant laboratory studies, made on the first day after his admission to the clinic fourteen days after the original onset of symptoms, are set down in table 7.

The diagnosis of hepatitis was rather obvious and surgical exploration was contraindicated. Whether this was a case of serum hepatitis or infectious hepatitis could not be determined. The patient's appetite rapidly improved and within two weeks the value for serum bilirubin had dropped decidedly. Six months later the man was well in every respect, and roentgenograms of the gallbladder revealed that it was functioning normally and did not contain stones.

Comment: Patients who have hepatitis tend to get well. The main concern of the physician then is correct diagnosis whereby he can avoid doing harm by radical measures.

CASE 6.—A white, male cotton ginner, 50 years of age, from Arkansas, was admitted because of "lack of pep" that had troubled him for nine months. His past history was essentially negative except that he had drunk a fifth (approximately 757 cc.) of whiskey each day for the past twenty years. Two and a half years previously, he said, he had had virus pneumonia and at that time, he reported, his local physician had felt a large liver that supposedly had decreased in size with treatment.

The present illness was of nine months' duration and had begun at a time when he "was drinking too much and eating too little." The course had been insidious but retrogressive. For six months prior to admission he had had intermittent jaundice, and it had been continuous for the previous four months. The urine had been intermittently dark, and the stools, on occasion, had been pale. His appetite had been poor and he had been weak and lacking in ambition. He had lost about 15 pounds (6.8 Kg.). On occasion he had had a temperature of 101 F. and night sweats had been frequent.

There had been mild "burning" of the feet but no true itching. Intermittently, his ankles had been slightly edematous. He complained that he bruised easily. Three weeks prior to his admission at the clinic he had been so fatigued that he had called on his physician, who had diagnosed hepatic disease. At no time did he complain of pain.

At the time of physical examination in Rochester, the man weighed 185 pounds (84 Kg.). His blood pressure was 190 mm. of mercury systolic and 110 diastolic. Two angiomatous spiders were present over the suprasternal notch. The palms were slightly red, fetor hepaticus was noticeable, and the man was moderately jaundiced. The liver extended 10 cm. below the costal margin and was slightly irregular to palpation. The spleen could not be felt, and there were no visible abdominal varices. No esophageal varices were visible through the esophagoscope. There was no ascites. Results of laboratory studies that were significant are recorded in table 8.

Rest; cessation of intake of all alcohol; increased intake of calories, carbohydrates, and proteins; and the taking of 1 hexavitamin capsule and 4 mg. of water-soluble vitamin K per day caused the patient's appetite to improve as did his general strength and sense of well-being. Within ten days the value for serum bilirubin had dropped to 3.5 mg. per 100 cc. direct and 1 mg. per 100 cc. indirect. The patient's temperature had been 100.5 F. during the first few days of his hospitalization but it became normal on the fifth day and remained within normal limits. He has gotten along well, and as this was written he was working eight hours a day.

Comment: The history of a large intake of alcohol may be significant here. Results of laboratory determinations were not very helpful. The prolonged prothrombin time, however, and its poor response to vitamin K indicated rather serious hepatocellular disease. Hepatic enlargement and the presence of jaundice definitely incriminated the liver. The finding of angiomatous spiders, the presence of fetor hepaticus and the fact that results of biopsy of the liver were consistent with severe cirrhosis with fatty changes clinched the diagnosis.

SUMMARY

In this paper the importance of a careful history, thorough physical examination, and confirmatory laboratory examinations in the differential diagnosis of mild, painless jaundice is emphasized. The problem of diagnosis is considered from two angles: (1) progressing from the evidence toward the diagnosis and (2) vice versa.

Illustrative case reports include patients with constitutional hepatic dysfunction, obstructive jaundice from a stone in the common bile duct, stricture of the common bile duct after surgery, biliary cirrhosis, infectious hepatitis, and portal cirrhosis.

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ABSTRACT OF DISCUSSION

DR. W. V. LEARY, Houston: I should like to emphasize a few of the points which Dr. Cain has made, which I am sure he would have emphasized had he had more time.

A casual perusal of the current literature might well lead one to the impression that numerous complicated laboratory procedures are necessary to establish the correct diagnosis. Such an impression has no basis in fact, and as the speaker has pointed out, approximately 75 per cent of cases can be correctly diagnosed if the physician will take the time to listen to the patient relate his history, ask certain pertinent questions, and carefully examine the patient. Having arrived at this impression, corroboration usually may be obtained by the use of a few simple laboratory tests rather than a battery of difficult procedures requiring highly trained technical personnel.

A distended gallbladder may be the key to the diagnosis in a given case of jaundice but usually requires meticulous physical examination for detection. Although rather deep palpation may be required to detect the distended organ, the examining hand must be gentle. The rough examiner who leaves the patient's abdomen covered with fingernail marks is not likely to palpate a gallbladder. A maneuver I have found useful is to have the patient tuck his right forearm behind his back at approximately the level of the liver, thus thrusting the contents of the right upper quadrant of the abdomen forward against the examiner's hand. On more than one occasion I have been able to palpate a gallbladder by this maneuver, whereas I had not been aware of its presence previously. This examining procedure may be particularly helpful when the patient is lying on a soft mattress and any pressure on the abdomen seems to force the various abdominal organs away from the examining hand. The same maneuver of tucking the left forearm behind the back may be used on the opposite side to palpate a spleen which previously had been impalpable.

It should be noted that certain of the laboratory procedures, although helpful in the diagnosis of hepatocellular disease, are actually not tests of liver function. These are the cephalin-cholesterol flocculation test, the thymol turbidity test, and the Kunkel test for gamma globulin. In the convalescent period of infectious hepatitis, one must have some guide in order properly to advise the patient with regard to resumption of activity. Since the three tests noted bear little or no relation to liver function, it is not reasonable to use them as a guide, particularly since they may remain positive for periods of more than a year. It is my own personal preference to rely on the bromsulfalein test and to permit full resumption of activity when this has returned to normal.

One of the tests which may be extremely useful and which is frequently neglected when one is concerned about the possibility of complete biliary obstruction is that of duodenal drainage. The quantitative determination of urobilinogen in a 72-hour stool and urine specimen as devised by Watson is undoubtedly one of the most reliable and useful tests in separating complete from incomplete biliary obstruction, as well as in other problems in the differential diagnosis of jaundice. However, it suffers from the disadvantage that it is a difficult test to perform and requires highly trained and skilled technical assistance. Duodenal drainage, on the other hand, is a relatively simple test and should be employed

more frequently, at least as a preliminary procedure, when the question of complete biliary obstruction arises. If equivocal results are obtained or confirmation seems desirable, the Watson test may then be used.

In general, the idea of promiscuous use of needle biopsies of vital organs does not appeal to me. In the case of the liver, though, this procedure may be added to the list of those which combine a high degree of reliability and a low degree of hazard. However, as with any procedure, needle biopsy of the liver will reach its greatest usefulness in the hands of those who are familiar with its limitations and contraindications. Dr. Cain has referred to the limitations. The chief contraindications are a low platelet count, prothrombin deficiency, peritonitis, subphrenic or hepatic abscess, severe ascites, and any evidence of disease involving the right pleural space.

WERNER'S SYNDROME
A Study of Adrenocortical and Hepatic Steroidal Metabolism

JERE M. BAUER and JEROME W. CONN, Ann Arbor, Michigan

IN 1904 Otto Werner[16] reported on the occurrence of "Cataracts in Connection with Scleroderma," an association which he had observed in four siblings. Thirty years later, Oppenheimer and Kugel[10] reported the first 2 such cases in the American literature. They pointed out the essential clinical features of a condition which they termed Werner's syndrome. Classic descriptions of this disease are available in these early reports as well as in two subsequent ones.[11, 15] In 1945 Thannhauser[15] was able to collect but 25 definite cases from the world's literature. Histories of these cases, however, suggested that the disease had occurred in either its complete form or in "forme fruste" in other members of the same families.

In its fully developed form the disease presents the following characteristics: shortness of stature with characteristic habitus, canities, premature baldness, scleropoikeloderma, trophic ulcers of the legs, juvenile cataracts, hypogonadism, tendency to diabetes mellitus, calcification of the blood vessels, osteoporosis, metastatic calcification, characteristic changes in larynx, and tendency to occur in siblings.

The present communication deals with data obtained in the course of a careful metabolic balance study upon a typical case of Werner's syndrome.

CASE REPORT

Mrs. E. C. was 44 years of age when first seen at the University Hospital. She complained chiefly of ulcerations on the feet. This, however, proved to be only one of a series of interesting events in her medical history. She had continued to grow until about 18 years of age, but her adult height was only 4 feet 8 inches. At the age of 12 years she first noted slight graying of the hair of the scalp, and this became complete at the age of 28 years. When she was 18 years old, deformities of the toes developed; these had progressed to a stage of severe contractures with overlapping of some of the toes and immobility of the phalanges. At the age of 28 years she developed cataracts which had necessitated bilateral lens extraction and ultimate removal of the left eye as a result of operative complications. At 32 years of age her menses became irregular and occurred only two or three times each year thereafter. Prior to this they had been normal since menarche at age 12 years. She had married at age 42 and had not been pregnant. When she was about 35 years old, she first noted a change in the voice, which at the time of examination was hoarse and squeaky. At the age of 41 years she developed the first cutaneous ulceration over the left achilles tendon. This was indolent and did not heal until skin grafted. Subsequently other ulcerations had developed on both ankles and about the lateral aspects of the feet. The patient had never grown much axillary or pubic hair and had noted no recent loss of hair in these areas. There had been considerable thinning of scalp hair, but the patient felt that there was continuing new growth of hair in this area. Pyorrhea alveolaris and loss of many teeth had occurred in the previous three years. The patient's weight usually was 75 pounds but had varied between 68 and 83 pounds. Whenever she gained weight, it was always distributed about the trunk.

One of the patient's sisters had died at age 63 years with an identical clinical picture. She was shorter than the patient, had developed gray hair at age 30 years, cataracts at 45 years, and had scant body hair and a squeaky voice. Another sister was of normal height and at age 63 years was obese and had diabetes mellitus and cataracts. There was no evidence of illness similar to the patient's in the other 5 siblings or in the parents.

The physical examination revealed a noticeably short woman who had an unusual body configuration (fig. 1a). The extremities were thin because of loss of subcutaneous fat and atrophy of the muscles, while the trunk was disproportionately large with a protuberant abdomen. The general appearance was that of a person much older than the patient's chronological age of 44 years. The face was rather sharp and the nose pointed. The scalp hair was steel gray and showed considerable thinning in the frontal and parietal areas. The hair on the extremities was sparse, and the axillary hair was decreased to about one-third of that usually

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expected for an adult female. The skin of the entire body had a thin, atrophic appearance with telangiectatic areas over the cheeks. The skin of the lower one-half of the legs and of the feet was indurated and bound down (fig. 1b). It had a shiny appearance and a waxy feel. In these regions there were intermingled areas of tan pigmentation and depigmentation. The skin over the lower parts of the legs and feet was so tight that movement at the ankles was limited to 50 per cent of normal and the metatarsal and interphalangeal joints were rigid. Some of the toes were drawn into an overlapping position. There were ulcers over the right lateral malleolus and at the base of the fifth metatarsal bone. A previous ulcer over the left achilles tendon had been successfully skin grafted. No arterial pulsations could be felt in the feet.

Other significant findings included a blood pressure of 154/84. There was irregularity of the right pupil following cataract extraction and a left ocular prosthesis. The breasts were normal. The liver was firm and descended 5 cm. below the right costal margin on deep inspiration. All tendon reflexes were greatly increased. Laryngoscopy showed the epiglottis to be prominent and elongated. The entire larynx was atrophic. The vocal cords were widely patent, and the laryngeal ventricles were visible. The cords approximated well, and the mucosa over the medial edge was clear and shiny. Beginning about 1 mm. from the medial edges of the cords there was a fine network of superficial, branching, injected blood vessels that passed across the cords into the ventricles. This duplicates the laryngeal findings of Oppenheimer and Kugel.[11]

Routine laboratory findings were within normal limits except for a white blood cell count of 10,350 and a sedimentation rate of 34 mm. per hour. A roentgenogram of the left ankle taken two years before admission to the University Hospital showed generalized osteoporosis and calcification of the vessels in the area. An electrocardiogram was normal. Urinary estrogen excretion was 20 rat units per twenty-four hours.

The etiology of Werner's syndrome is not known, though the heredofamilial features indicate a genetic factor. In addition, an endocrine origin for the disorder has been suggested. When the available information is reviewed critically, however, one finds no evidence of endocrine dysfunction that could play a primary etiologic role in this disease. Although many of these patients exhibit both diabetes mellitus and hypogonadism, neither of these defects can account for the profound somatic changes observed in this disease. Increased function of the parathyroid glands was proposed by Oppenheimer and Kugel,[10, 11] but the evidence for this is not convincing. Because several clinical features of Werner's syndrome suggest the possibility of an unusual form of hyperfunction of the adrenal cortex, we have studied the adrenocortical physiology of the case just reported.

FIG. 1a. Photograph of a 44 year old woman with Werner's syndrome, showing thin extremities and a disproportionately large trunk with protuberant abdomen.

b. Feet and legs of the patient. The skin was indurated and bound down, waxy and shiny, and had intermingled areas of depigmentation and tan pigmentation.

METHOD OF STUDY

The patient was fed a constant weighed diet containing 54.5 Gm. of protein, 156.0 Gm. of carbohydrate, 97.8 Gm. of fat, 3,148 mg. of sodium, 4,897 mg. of chloride, and 2,647 mg. of potassium. The first six days established a reliable baseline. During the next six days the patient was given 70 mg. of ACTH intramuscularly daily in 3 divided doses. The following five days served as a basis for study of the recovery period after ACTH administration.

The determinations performed were blood sugar,[9] serum cholesterol and cholesterol esters,[14] urinary uric acid,[1, 2] glucose,[9] chlorides,[8] 17-ketosteroids,[13] formaldehydogenic steroids,[5] quantitative blood eosinophil counts,[12] and blood hematocrit level.[17] All nitrogen determinations were done by the macro-Kjeldahl method. Glucose tolerance tests were done by administering orally 2 Gm. of glucose per kilogram of body weight.

RESULTS

The patient experienced no untoward symptoms while ACTH was being administered. During this period there was a gain of 3 pounds in body weight and the blood pressure increased to 178/84. On the third day of administration of ACTH she noted increase in the mobility of the ankle joints. On the fourth day of ACTH administration the dilated vessels on the cheeks were more prominent. On this day she menstruated for the first time in several months. On the fifth day of administration of ACTH the wound of a skin biopsy made ten days earlier broke open.

The general metabolic responses to ACTH shown in this case of Werner's syndrome were similar to those seen in normal persons. However, an unusual pattern of steroid excretion was encountered.

Figure 2 shows the effect of ACTH upon the levels of blood sugar; on urinary sugar, nitrogen, and uric acid; and on the circulating eosinophils. In the control period the fasting blood sugar level was at the upper limits of normal.* During the six days of ACTH administration there was a sustained elevation of the fasting blood sugar level. On the last day of ACTH administration it reached 137 mg. per 100 cc. Within twenty-four hours after stopping the ACTH the fasting blood sugar value was 70 mg. per 100 cc., and thereafter it gradually returned to levels at, or slightly above, those found in the control period. The urinary excretion of glucose paralleled closely the changes in the fasting blood sugar. Significant glycosuria (that is, excretion of glucose greater than 1 Gm. per day) occurred on only one day of the control period. During the period of ACTH administration, however, there was a constant glycosuria varying between 9 and 19 Gm. of glucose per day. During the recovery period significant glycosuria occurred only on the first day after stopping the ACTH.

Urinary nitrogen excretion during the control period was somewhat less than would be expected for equilibrium, indicating a slight positive nitrogen balance. When ACTH was administered, the urinary nitrogen excretion showed a progressive increase and the patient was in constant negative nitrogen balance. On the sixth day of ACTH administration this negative nitrogen balance reached a value of 3.13 Gm. During the recovery period the urinary nitrogen excretion returned toward the level expected for nitrogen balance.

The urinary excretion of uric acid was within the normal range in the control period. When ACTH was

*Normal range of fasting blood sugar, 70 to 100 mg. per 100 cc.

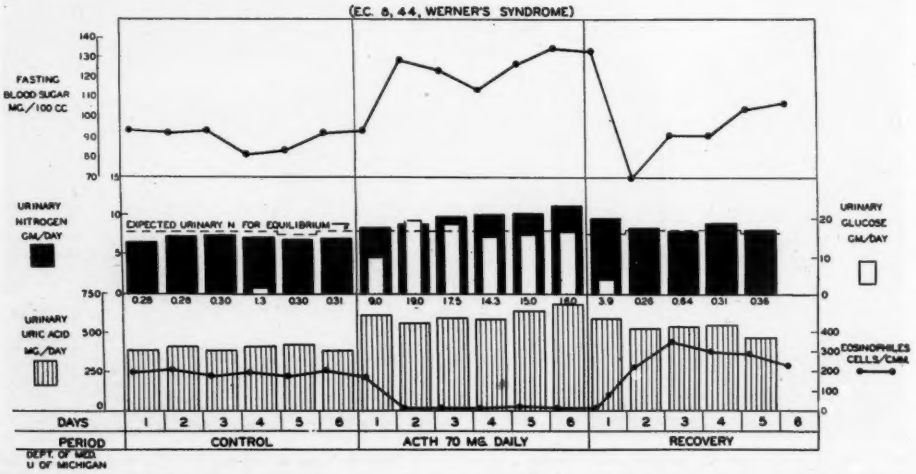
(EC. 8, 44, WERNER'S SYNDROME)
FASTING BLOOD SUGAR MG./100 CC
URINARY NITROGEN GM/DAY
EXPECTED URINARY N FOR EQUILIBRIUM
URINARY GLUCOSE GM/DAY
URINARY URIC ACID MG./DAY
EOSINOPHILES CELLS/CMM.
0.28 0.28 0.30 1.3 0.30 0.31 9.0 19.0 17.5 14.3 15.0 16.0 3.9 0.26 0.64 0.31 0.36
DAYS 1 2 3 4 5 6 1 2 3 4 5 6 1 2 3 4 5 6
PERIOD
CONTROL
ACTH 70 MG. DAILY
RECOVERY
DEPT. OF MED. U OF MICHIGAN

FIG. 2. Graph showing the effect of ACTH upon blood sugar levels and upon urinary sugar, nitrogen, and uric acid.

given there was a definite increase in the daily excretion of uric acid. On cessation of ACTH administration the uric acid excretion gradually declined.

The level of circulating eosinophils was normal during the control period. The expected disappearance of eosinophils was seen during ACTH administration, and there was an immediate rebound in the eosinophil level after stopping the ACTH.

Figure 3 shows the results of glucose tolerance tests done before ACTH was given (curve 1), after six days of ACTH administration (curve 2), and five days after stopping ACTH (curve 3). It will be noted that even before ACTH was given, the curve was typically diabetic, with a normal fasting blood sugar level. During ACTH administration, carbohydrate tolerance was depressed further and the fasting blood sugar level rose. Five days after the last dose of ACTH, carbohydrate tolerance was still somewhat below the control level.

Figure 4 shows the expected response of the total, esterified, and free cholesterol of serum to the administration of ACTH.[4] During the control period the total cholesterol level and its distribution between the free and esterified forms was normal. When ACTH was given, there was a gradual decrease in both the esterified and free cholesterol, the greatest percentage decrease occurring in the ester fraction. By the sixth day of the recovery period both the esterified and free cholesterol had returned to the control values.

Figure 5 shows the effect of ACTH upon the body weight, the urinary chloride excretion, and the hematocrit level. Fluid retention during the period of ACTH administration is indicated by a gain in body weight of 3 pounds and a decrease in the hematocrit level from 39 per cent to 32.9 per cent in six days. When ACTH was stopped, there was a prompt loss in body weight and an increase in the hematocrit to the control level. During the first four days of ACTH administration, retention of chloride occurred. During the last two days of the ACTH period, however, there occurred a diuresis of chloride, a phenomenon known to occur in most normal people.

Figure 6 shows the pattern of excretion of urinary 17-ketosteroids and of formaldehydogenic steroids. In

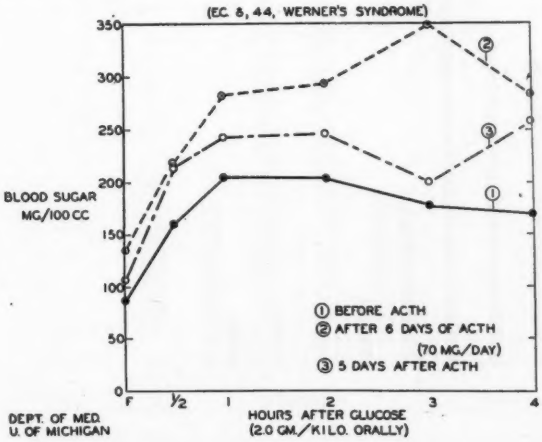
(EC. 8, 44, WERNER'S SYNDROME)
BLOOD SUGAR MG/100 CC
① BEFORE ACTH
② AFTER 6 DAYS OF ACTH (70 MG./DAY)
③ 5 DAYS AFTER ACTH
HOURS AFTER GLUCOSE (2.0 GM./KILO. ORALLY)
DEPT. OF MED. U. OF MICHIGAN

FIG. 3. The effect of ACTH upon glucose tolerance.

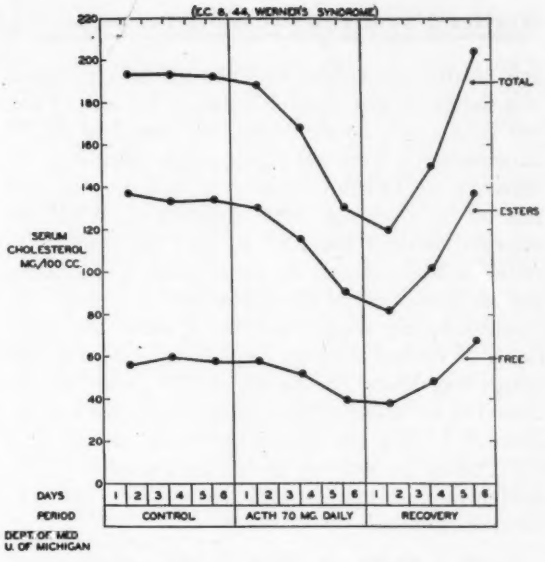

FIG. 4. The effect of ACTH upon serum cholesterol.

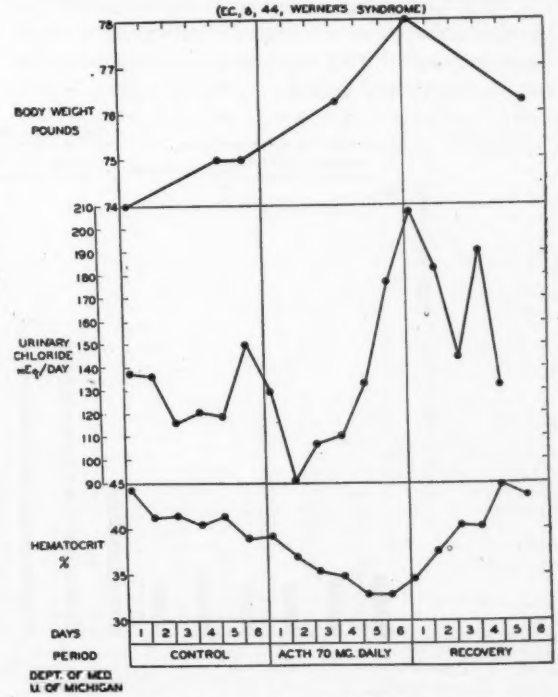

FIG. 5. The effect of ACTH upon body weight, urinary chloride excretion, and hematocrit.

the control period the excretion of 17-ketosteroids was somewhat low, ranging between 5.5 and 7.2 mg. per twenty-four hours. During the period of ACTH administration there was a progressive increase in the excretion of 17-ketosteroids to a maximum of 47.8 mg. on the sixth day. After cessation of ACTH administration there was a fall in the 17-ketosteroid excretion within a day to the levels found in the control period. In the control period the level of urinary formaldehydogenic steroids was at or above the upper limits of normal, ranging from 0.92 to 2.6 mg. per twenty-four hours. During the ACTH period the excretion of formaldehydogenic steroids reached a maximum of 4.9 mg. per twenty-four hours on the fifth day. During the recovery period the excretion of formaldehydogenic steroids decreased to levels found in the control period.

Figure 7 shows the effect of orally administered free compound S on the urinary excretion of 17-ketosteroids. This test was conducted thirty-two months after the previous studies with ACTH. It will be noted that in the control period the urinary excretion of 17-ketosteroids (3.9 and 4.3 mg. per day) is somewhat lower than that observed thirty-two months previously. When 400 mg. of free compound S was given orally for two days, 17-ketosteroid excretion increased to 11.1 mg. the first day and 18.3 mg. the second day. This 17-ketosteroid excretion on the second day of administration of compound S represents a maximum increase of 14.2 mg. above the average excretion for the control period (4.1 mg. per day). This is a considerably smaller increase than that which occurs in normal persons.[7] Five days after compound S had been stopped the 17-ketosteroid excretion was again 4.3 mg. per day. The excretion of formaldehydogenic steroids at this time was 3 mg. per twenty-four hours.

When liver function was tested by conventional means the values for all tests were normal; total serum bilirubin 0.6 mg. per 100 cc., free bilirubin (1 minute) 0, free bilirubin (direct thirty minute) 0.2 mg. per 100 cc., combined bilirubin 0.4 mg. per 100 cc., gamma globulin 7.7 units, thymol turbidity 1.9 units, cephalin flocculation 0 at twenty-four and forty-eight hours, total serum cholesterol 202 mg. per 100 cc. with 69 per cent of the total in the esterified form.

COMMENT

Figures 2 through 6 indicate that the adrenocortical as well as the metabolic responses to ACTH were normal in this case of Werner's syndrome. Of particular interest, however, is the unusual relationship to each other of excretion of 17-ketosteroid and formaldehydogenic steroids. This pattern consists of subnormal excretion of 17-ketosteroids in association with elevated values for formaldehydogenic steroids (fig. 6) in both the control and recovery periods. Since this type of dissociation in the pattern of excretory steroids is observed in hepatic disease, the experiment involving the ingestion of compound S was added to the program.

We have reported that oral administration of compound S, a relatively inert steroid from the metabolic point of view, results in a sharp increase in urinary 17-ketosteroids when the test is performed on persons

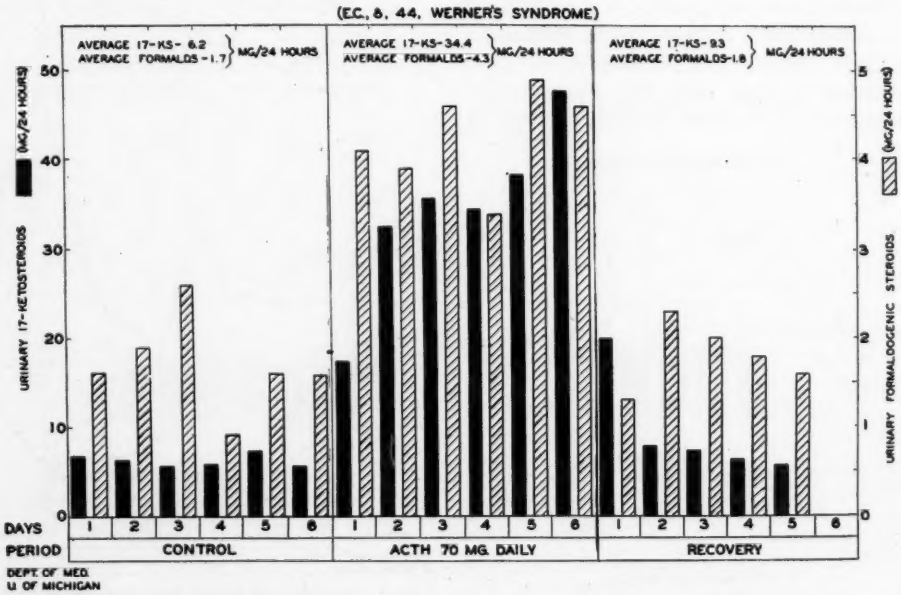

FIG. 6. The effect of ACTH upon urinary excretion of 17-ketosteroids and formaldehydogenic steroids.

with normal liver function.[7] On the other hand, when the same procedure is carried out in patients with hepatic disease, a much smaller increase in 17-ketosteroid excretion occurs.[6] The response obtained in this patient with Werner's syndrome is similar to that found in patients with severe hepatic dysfunction. Although moderate hepatomegaly is present, conventional tests of liver function have disclosed no abnormalities. On the basis of our experience with compound S to date,[3] the result obtained in the present patient is highly suggestive of an inability of the liver to facilitate the conversion of compound S to excretory 17-ketosteroids.

Since compound F (hydrocortisone) is similarly converted by the liver to excretory 17-ketosteroids,[6] it is probable that the mild elevation in urinary formaldehydogenic steroids associated with mild depression of 17-ketosteroid excretion represents a disturbance in hepatic transformation of endogenously produced 17-hydroxycorticoids to excretory 17-ketosteroids. This situation does not imply necessarily any abnormality in the secretory activity of the adrenal cortex.

A fundamental disturbance from birth in the capacity of the liver to carry out certain transformations of physiologically active adrenal steroids to relatively inactive excretory products conceivably could account for many of the clinical manifestations of the fully developed picture of Werner's syndrome. In this sense, the manifestations would be regarded as being the result of long continued, mildly excessive metabolic activities of steroids of the compound F type, produced in normal quantity, but excessive because of failure of the liver to degrade them with sufficient rapidity. Thus, failure to grow and to develop normally, the tendency to diabetes mellitus, the osteoporosis, and the evidences of tissue atrophy all suggest an antianabolic influence of long duration. Whereas one could argue justifiably that the end result of such a process should be full blown Cushing's syndrome, it is likely that modifying influences such as onset at or before birth and mild degree and long duration of excessive activity of steroids of the 11-17-oxygenated type can determine the final clinical manifestations. If this aberration in the hepatic metabolism of steroid compounds can be confirmed in other cases of this rare syndrome, the situation might then fall into the group of the so-called "inborn errors of metabolism."

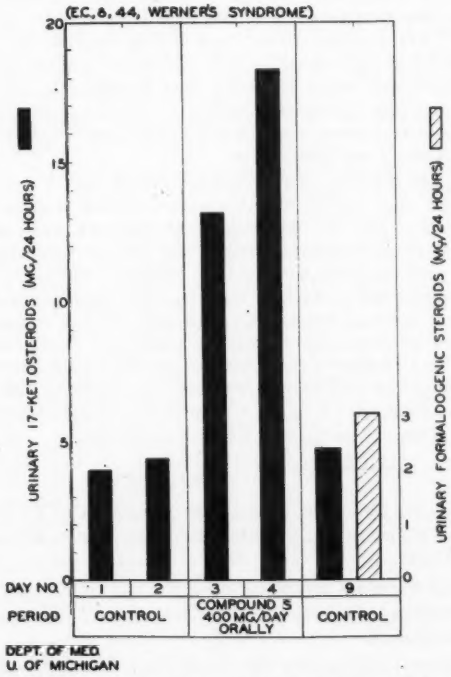

FIG. 7. The effect of orally administered compound S on the urinary excretion of 17-ketosteroids.

CONCLUSIONS

Balance studies carried out upon a well documented case of Werner's syndrome revealed normal metabolic and urinary steroidal responses to administration of ACTH. However, in the absence of adrenocortical stimulation with exogenous ACTH, excretion of 17-ketosteroids was subnormal, while excretion of formaldehydogenic steroids was elevated.

Administration of compound S disclosed deficiency in hepatic conversion of this compound to excretory 17-ketosteroids, despite the absence of other indications of abnormal liver function.

It is suggested that Werner's syndrome may consist primarily of an aberration in the hepatic metabolism of steroidal compounds.

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12. Randolph, T. G.: Differentiation and Enumeration of Eosinophils in Counting Chamber with Glycol Stain; Valuable Technique in Appraising ACTH Dosage, J. Lab. & Clin. Med. *34*:1696-1701 (Dec.) 1949.

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14. Sperry, W. M.: Schoenheimer-Sperry Method for Determination of Cholesterol, Revised, New York, Department of Biochemistry, New York Psychiatric Institute and Hospital, April, 1945.

15. Thannhauser, S. J.: Werner's Syndrome (Progeria of Adult) and Rothmund's Syndrome; Two Types of Closely Related Heredofamilial Atrophic Dermatoses with Juvenile Cataracts and Endocrine Features; Critical Study with Five New Cases, Ann. Int. Med. *23*:559-626 (Oct.) 1945.

16. Werner, C. W. O.: Ueber Katarakt in Verbindung mit Sklerodermie, Inaug. Diss. Kiel, Schmidt & Klaunig, 1904.

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ABSTRACT OF DISCUSSION

DR. MAVIS P. KELSEY, Houston: I have never seen a patient with Werner's syndrome, and I know little about the metabolism of steroids. Perhaps this ignorance has led me to the audacity of asking a few questions. (1) Although Werner's syndrome has some features in common with Cushing's syndrome, is it not far removed from Cushing's syndrome as a clinical entity? (2) Is or is not the subnormal increase of 17-ketosteroid excretion after the administration of compound S and the slight discrepancy between the excretory levels of 17-ketosteroids and the formaldehydogenic steroids in this one patient rather slim evidence of an inborn error of metabolism? (3) Is the lack of other evidence of hepatic insufficiency in this patient against Dr. Bauer's theory? (4) Should ACTH aggravate, not improve, this condition if it is apparently due to hyperadrenocorticism?

In spite of these questions, I think this is a brilliant theory: that Werner's syndrome is the result of the inability of the liver to metabolize steroids of compound F type and thus cause a low grade hypercorticism. Dr. Bauer's discussions and metabolic studies certainly answer in part the questions I have raised and his theory certainly comes more closely to explaining the syndrome than does either the theory of hyperparathyroidism, hypogonadism, or congenital weakness of the ectoderm. Along this line of thinking, there are some bits of information which may be significant. For example, the only reported patient with Werner's syndrome who came to autopsy had large adrenal glands with multiple adenomas of the cortex. The administration of corticosteroids to chick embryos stunts their development and produces enlarged eyes, which is an unusual parallel to the stunted growth and cataracts seen in Werner's syndrome.

It may be important that several relationships exist between the liver and spleen, even though superficial evaluation shows no apparent clues to Dr. Bauer's theory. Conn and other workers have shown that the adrenal cortex seems to take cholesterol from the blood stream to synthesize cortical hormones, this cholesterol being released into the blood stream by the liver. There is also the complex interrelationship of the adrenal cortex and the liver involved in the metabolism of proteins and carbohydrates. There is diminished excretion of 17-ketosteroids with liver disease and there is diminished liver function with Addison's disease. In cirrhosis in males, there may be gynecomastia and atrophy of the testes, apparently the result of retention of estrogens secreted by the adrenal cortex and not metabolized by the damaged liver. Striking clinical improvement has been reported in cirrhosis and hepatitis following the administration of cortisone.

DR. BAUER, closing: I would like to thank Dr. Kelsey for his comments and questions, to which I will reply in order.

1. It is true that in its gross clinical appearance, Werner's syndrome seems to lack many of the characteristics that we associate with Cushing's syndrome. Examined more closely, however, there are several important points of similarity, namely, the diabetes mellitus, osteoporosis, and evidence of tissue atrophy. These features are those that one would expect with increased activity of adrenal steroids of the compound F variety. The differences in Werner's syndrome and Cushing's syndrome may be due in the case of the former disease to the early age of onset and the mild degree and long duration of excessive activity of adrenal steroids of the 11-17-oxygenated type.

2. The findings in 1 case of anything constitute slim evidence on which to make a final judgment. We have reported these studies of adrenocortical and hepatic steroidal metabolism in this case to stimulate similar studies in other cases of this rare disease. If the abnormality in hepatic steroidal metabolism found in this case can be confirmed in other patients with the disease, it would suggest that it might be classified in the group of "inborn errors of metabolism."

3. I do not believe that the lack of other evidence of hepatic insufficiency in this patient negates the interpretations that have been made. Our experience thus far with the use of oral compound S as a test of liver function has shown it to be a more sensitive test than those usually used. In addition, this means of testing liver function has the virtue of specificity in that it is testing the ability of the liver to metabolize adrenal steroids, a point of primary interest in the study of the present case.

4. On the basis of our findings in this case, I would anticipate that ACTH would aggravate rather than improve Werner's syndrome. Our studies in this case were not designed for therapeutic purposes; they were of short duration and were done only to record the metabolic effect of ACTH. I am inclined to believe that the slight improvement the patient reported was largely subjective and was due in part to the usual psychic stimulation seen with ACTH and in part to a feeling of optimism on her part, since even after stopping the ACTH she felt that she remained somewhat better. I certainly would not recommend ACTH or cortisone as a form of therapy for Werner's syndrome.

Midwinter Seminar in Florida

The eighth annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held January 18-23 in Miami Beach. Lectures on ophthalmology will be presented during the first three days of the seminar by Dr. W. B. Anderson, Durham, N. C.; Dr. W. P. Beetham, Boston; Dr. W. C. Owens, Baltimore; and Drs. A. B. Reese and M. C. Wheeler, New York. During the last three days lectures on otolaryngology will be given by Dr. E. N. Broyles, Baltimore; Dr. H. P. House, Los Angeles; Dr. W. J. McNally, Montreal; and Drs. Dorothy Wolff and D. Woodman, New York.

Registrants are invited to attend the midwinter convention of the Florida Society of Ophthalmology and Otolaryngology on January 20.

Further information may be obtained from Dr. Shaler Richardson, Graduate School of Medicine, University of Florida, Jacksonville.

NEWS

COMING MEETINGS AND CLINICS

Texas Medical Association, San Antonio, May 3-5, 1954. Dr. George Turner, El Paso, Pres.; Mr. N. C. Forrester, 1801 Lamar Blvd., Austin, Executive Secy.

American Medical Association, St. Louis, Dec. 1-4, 1953. Dr. Edward J. McCormick, Toledo, Ohio, Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy, Houston, Feb. 1-3, 1954. Dr. Ben Z. Rappaport, Chicago, Pres.; Dr. Alan G. Cazort, 1425 West 7th St., Little Rock, Secy.

American Academy of Dermatology and Syphilology, Chicago, Dec. 5-10, 1953. Dr. Michael H. Ebert, Chicago, Pres.; Dr. John E. Rauschkolb, 25 Prospect Ave., N W., Cleveland 15, Secy.

American Academy of General Practice. Dr. R. B. Robins, Camden, Ark., Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Secy.

American Academy of Obstetrics and Gynecology, Cincinnati, Dec. 14-16, 1953. Dr. Robert A. Kimbrough, Jr., Philadelphia, Pres.; Dr. C. Paul Hodgkinson, Detroit, Secy.

American Academy of Ophthalmology and Otolaryngology. Dr. Frederick C. Cordes, San Francisco, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics. Dr. Phillip Barba, Philadelphia, Pres.; Dr. E. H. Christopherson, 610 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery, Montreal, Canada, May 3-5, 1954. Dr. Emile F. Holman, San Francisco, Pres.; Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif., Secy.

American Association of Genito-Urinary Surgeons, Shawnee-on Delaware, Pa., May 26-28, 1954. Dr. Vincent J. O'Conor, Chicago, Pres.; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago 3, Secy.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Hot Springs, Va., Sept. 9-11, 1954. Dr. Herbert F. Schmitz, Chicago, Pres.; Dr. F. R. Lock, Bowman Gray School, Winston-Salem, N. C., Secy.

American Cancer Society. Dr. Guy Aud, Louisville, Ky., Pres.; Mr. M. R. Runyon, 47 Beaver St., New York, Executive Vice-Pres.

American College of Allergists, Miami Beach, April 5-10, 1954. Dr. M. Murray Peshkin, New York, Pres.; Dr. Fred W. Wittich, 423 La Salle Medical Bldg., Minneapolis 2, Secy.

American College of Chest Physicians, San Francisco, June 17-20, 1954. Dr. Alvis E. Greer, Houston, Pres.; Mr. Murray Kornfeld, 112 E. Chestnut St., Chicago 11, Executive Secy.

American College of Physicians, Chicago, April 5-9, 1954. Dr. Leroy H. Sloan, Chicago, Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology, Chicago, February, 1954. Dr. John D. Camp, Los Angeles, Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secy.

American College of Surgeons. Dr. Harold L. Foss, Danville, Pa., Pres.; Dr. Michael L. Mason, 40 E. Erie St., Chicago 11, Secy.

American Congress of Physical Medicine and Rehabilitation, Washington, D. C., Sept. 6-11, 1954. Dr. William B. Snow, New York, Pres.; Dr. Frances Baker, 1 Tilton Ave., San Mateo, Calif., Secy.

American Dermatological Association, White Sulphur Springs, W. Va., April 13-17, 1954. Dr. Louis A. Brunsting, Rochester, Minn., Pres.; Dr. J. Lamar Callaway, Duke Hospital, Durham, N. C., Secy.

American Gastro-Enterological Association, San Francisco, June 18-19, 1954. Dr. J. M. Ruffin, Durham, N. C., Pres.; Dr. H. Marvin Pollard, 1313 E. Ann St., Ann Arbor, Mich., Secy.

American Gynecological Society, Hot Springs, Va., May 20-22, 1954. Dr. Richard W. TeLinde, Baltimore, Pres.; Dr. John I. Brewer, 104 S. Michigan Ave., Chicago, Secy.

American Heart Association, Chicago, April 1-4, 1954. Dr. Robert L. King, Seattle, Pres.; Dr. William H. Bunn, 44 E. 23d St., New York 10, Secy.

American Hospital Association. Dr. Edwin L. Crosby, Chicago, Pres.; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secy.

American Laryngological, Rhinological, and Otological Society, Boston, May 25-27, 1954. Dr. LeRoy A. Schall, Boston, Pres.; Dr. C. S. Nash, 277 Alexander St., Rochester 7, N. Y., Secy.

American Neurological Association, Atlantic City, N. J., June 14-16, 1954. Dr. Roland P. Mackay, Chicago, Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, Glacier Park, Mont., June 16-18, 1954. Dr. William L. Benedict, Rochester, Minn., Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association, Bretton Woods, N. H., June 6-9, 1954. Dr. A. R. Shands, Jr., Wilmington, Del., Pres.; Dr. George O. Eaton, 4 E. Madison St., Baltimore 2, Secy.

American Pediatric Society, Buck Hill Falls, Pa., May 3-5, 1954. Dr. Rustin McIntosh, New York, Pres.; Dr. Aims McGuinness, 237 Medical Laboratory, University of Pennsylvania, Philadelphia 46, Secy.

American Proctologic Society, Los Angeles, June 2-5, 1954. Dr. Wendell Green, Toledo, Ohio, Pres.; Dr. Stuart T. Ross, 131 Fulton Ave., Hempstead, N. Y., Secy.

American Psychiatric Association, St. Louis, May 3-7, 1954. Dr. Kenneth E. Appel, Philadelphia, Pres.; Dr. R. Finley Gayle, 6300 Three Chopt Rd., Richmond 21, Va., Secy.

American Public Health Association. Dr. Wilton L. Halverson, San Francisco, Pres.; Dr. R. W. Atwater, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists. Dr. Ralph T. Knight, Minneapolis, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists, Washington, D. C., Sept. 6, 1954. Dr. John R. Schencken, Omaha, Pres.; Dr. Clyde G. Culbertson, Indiana University School of Medicine, Indianapolis, Secy.

American Surgical Association, Cleveland, April, 1954. Dr. Howard C. Naffziger, San Francisco, Pres.; Dr. R. Kennedy Gilchrist, 59 East Madison St., Chicago 3, Secy.

American Urological Association, New York, May 31-June 3, 1954. Dr. J. A. Campbell Colston, Baltimore, Md., Pres.; Dr. C. H. deT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

Association of American Physicians and Surgeons, Jackson, Miss., April 1-3, 1954. Dr. Thomas G. Goldsmith, Greenville, S. C., Pres.; Mr. Harry E. Northam, 360 N. Michigan Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter, Chicago, Sept. 7-10, 1954. Dr. William R. Lovelace, Albuquerque, N. M., Pres.; Dr. Karl Meyer, 1516 Lake Shore Drive, Chicago, Secy.

National Tuberculosis Association, Atlantic City, N. J., May 17-21, 1954. Mr. Mark H. Harrington, Denver, Pres.; Mr. Kemp D. Battle, 1790 Broadway, New York 19, Secy.

Radiological Society of North America, Chicago, Dec. 13-18, 1953. Dr. Ira H. Lockwood, Kansas City, Mo., Pres.; Dr. D. S. Childs, 713 E. Genesee, Syracuse 2, N. Y., Secy.

Southern Medical Association, St. Louis, 1954. Dr. Alphonse McMahon, St. Louis, Pres.; Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham 3, Ala., Secy.

Southern Psychiatric Association, Louisville, October, 1954. Dr. John D. Trawick, Louisville, Ky., Pres.; Dr. Joseph L. Knapp, 210 N. Westmoreland, Dallas, Secy.

Southern Surgical Association, Hot Springs, Va., Dec. 8-10, 1953. Dr. I. A. Bigger, Richmond, Va., Pres.; Dr. John C. Burch, 2112 West End Ave., Nashville 5, Tenn., Secy.

Southwest Allergy Forum, New Orleans, May 9-11, 1954. Dr. Henry D. Ogden, New Orleans, Pres.; Dr. Stanley Cohen, 1441 Delachaise St., New Orleans, Secy.

Southwest Regional Cancer Conference. Secy., 209 Medical Arts Bldg., Fort Worth.

Southwestern Medical Association. Dr. Wesley O. Connor, Jr., Albuquerque, N. M., Pres.; Dr. Celso Stapp, 800 Montana, El Paso, Secy.

Southwestern Surgical Congress, Oklahoma City, Sept. 27-29, 1954. Dr. Philip B. Price, Salt Lake City, Pres.; Dr. C. R. Rountree, 1227 Classen, Oklahoma City 3, Secy.

Tri-State Medical Assembly, Fall, 1954. Dr. Joe D. Nichols, Atlanta, Pres.; Dr. Broox C. Garrett, 940 Margaret Pl., Shreveport, La., Secy.

United States-Mexico Border Public Health Association, Albuquerque, N. M., April, 1954. Mr. Richard F. Poston, Dallas, Pres.; Dr. Sidney B. Clark, 314 U. S. Court House, El Paso, Secy.

STATE

Private Clinics and Hospitals Association of Texas, Austin, Dec. 5-6, 1953. Dr. S. D. Coleman, Navasota, Pres.; Dr. Neil D. Buie, Marlin, Secy.

Texas Academy of General Practice. Dr. C. Forrest Jorns, Houston, Pres.; Dr. Woodson W. Harris, 1410 Nickerson, Austin, Secy.

Texas Academy of Internal Medicine, Houston, Dec. 5-6, 1953. Dr. Hatch W. Cummings, Jr., Houston, Pres.; Dr. George M. Jones, 1314 Medical Arts Bldg., Dallas, Secy. Meetings restricted to members.

Texas Air-Medics Association, San Antonio, May 2-3, 1954. Dr. George L. Gallaher, Harlingen, Pres.; Dr. J. S. Minnett, 2512 Welborn, Dallas, Secy.

Texas Association of Blood Banks, Fort Worth, Dec. 4-5, 1953. Dr. W. N. Powell, Temple, Pres.; Miss Majorie Saunders, 3500 Gaston Ave., Dallas, Secy.

Texas Association of Obstetricians and Gynecologists, Waco, Feb. 19-20, 1954. Dr. D. D. Wall, San Angelo, Pres.; Dr. Carey Hiett, 815 Fifth Ave., Fort Worth, Secy.

Texas Chapter, American College of Chest Physicians, San Antonio, May 2, 1954. Dr. Henry H. Hoskins, San Antonio, Pres.; Dr. Walter C. Brown, 1733 S. Brownlee Blvd., Corpus Christi, Secy.

Texas Club of Internists. Dr. Joseph F. McVeigh, Fort Worth, Pres.; Dr. Charles Darnall, Capital National Bank Bldg., Austin, Secy.

Texas Dermatological Society, San Antonio, May 2, 1954. Dr. Maurice Barnes, Waco, Pres.; Dr. Thomas L. Shields, 1216 Pennsylvania Ave., Fort Worth, Secy.

Texas Diabetes Association, San Antonio, May 2, 1954. Dr. Raymond L. Gregory, Galveston, Pres.; Dr. Edmond K. Doak, 501 Hermann Professional Bldg., Houston, Secy.

Texas Division, American Cancer Society, Austin, Jan. 14-15, 1954. Mr. Travis Wallace, Dallas, Pres.; Mr. J. Louis Neff, 1609 Colorado, Austin, Executive Director.

Texas Heart Association, San Antonio, May 3, 1954. Dr. Joseph McVeigh, Fort Worth, Pres.; Miss Roberta Miller, 412 Fidelity Union Life Bldg., 1511 Bryan St., Dallas, Executive Secy.

Texas Hospital Association, Houston, May 18-20, 1954. Mr. W. U. Paul, El Paso, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.

Texas Neuropsychiatric Association, San Antonio, May 2, 1954. Dr. James Blair, San Antonio, Pres.; Dr. John L. Otto, John Sealy Hospital, Galveston, Secy.

Texas Orthopedic Association, San Antonio, May 3, 1954. Dr. Edward T. Smith, Houston, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.

Texas Pediatric Society, Fort Worth, October, 1954. Dr. Robert L. Moore, Dallas, Pres.; Dr. James N. Walker, 3616 Tulsa Way, Fort Worth, Secy.

Texas Public Health Association, Galveston, Feb. 14-17, 1954. Dr. Roy G. Reed, La Marque, Pres.; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secy.

Texas Radiological Society, Dallas, Jan. 29-30, 1954. Dr. J. E. Miller, Dallas, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.

Texas Railway and Traumatic Surgical Association, San Antonio, May 3, 1954. Dr. William E. Crump, Wichita Falls, Pres.; Dr. W. D. Marrs, 306 Broadway, Fort Worth, Secy.

Texas Rheumatism Association, Houston, Dec. 4, 1953. Dr. J. C. Crager, Beaumont, Pres.; Dr. Charles H. Cornwell, Marlin, Secy.

Texas Society for Mental Health, Waco, March 4-6, 1954. Mrs. Frank Schoonover, Fort Worth, Pres.; Mrs. Elizabeth F. Gardner, 2504 Jarratt Ave., Austin 21, Executive Secy.

Texas Society of Anesthesiologists, San Antonio, May 2, 1954. Dr. James B. Robinett, Jr., Houston, Pres.; Dr. C. R. Allen, John Sealy Hospital, Galveston, Secy.

Texas Society of Gastroenterologists and Proctologists, San Antonio, May 3, 1954. Dr. Cecil O. Patterson, Dallas, Pres.; Dr. W. T. Arnold, 1402 Hermann Prof. Bldg., Houston, Secy.

Texas Society of Ophthalmology and Otolaryngology, Fort Worth, Dec. 4-5, 1953. Dr. Dan Brannin, Dallas, Pres.; Dr. Lyle Hooker, 627 Esperson Bldg., Houston, Secy.

Texas Society of Pathologists, Galveston, Jan. 31, 1954. Dr. A. O. Severance, San Antonio, Pres.; Dr. Lloyd R. Hershberger, Shannon Memorial Hospital, San Angelo, Secy.

Texas Surgical Society, Waco, April 5-6, 1954. Dr. Dudley Jackson, Sr., San Antonio, Pres.; Dr. Albert W. Hartman, 414 Navarro St., San Antonio 5, Secy.

Texas Tuberculosis Association, Dallas, April 9-10, 1954. Dr. R. G. McCorkle, San Antonio, Pres.; Miss Pansy Nichols, 208 E. Ninth, Austin, Executive Secy.

Texas Urological Society, Dr. J. Reese Blundell, Houston, Pres.; Dr. Dolphus E. Compere, 1415 Pennsylvania Ave., Fort Worth, Secy.

DISTRICT

First District Society, Pecos, Feb. 12, 1954. Dr. John W. O'Donnell, Alpine, Pres.; Dr. H. D. Garrett, First National Bldg., El Paso, Secy.

Second District Society, Sweetwater, April 15, 1954. Dr. T. D. Young, Sweetwater, Pres.; Dr. Francis Hood, Sweetwater, Secy.

Third District Society, Amarillo, April 13, 1954. Dr. Robert A. Neblett, Canyon, Pres.; Dr. James T. Hall, 1626 Fifteenth St., Lubbock, Secy.

Fourth District Society, San Angelo, Fall, 1954. Dr. P. M. Wheelis, Brownwood, Pres.; Dr. James N. White, San Angelo, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July, 1954. Dr. Y. C. Smith, Sr., Corpus Christi, Pres.; Dr. Robert J. Sigler, 1126 Third Street, Corpus Christi, Secy.

Seventh District Society, Austin, Feb. 18, 1954. Dr. Benjamin Clary Bates, Austin, Pres.; Dr. William McLean, 4618 Burnet Road, Austin, Secy.

Eighth District Society. Dr. Howard Z. Fretz, Wharton, Pres.; Dr. George E. Glover, Jr., Victoria, Secy.

Ninth District Society, Brenham, March 25, 1954. Dr. George D. Bruce, Baytown, Pres.; Dr. Lyman C. Blair, 1212 Rothwell, Houston, Secy.

Tenth District Medical Society. Dr. Edmund D. Jones, Beaumont, Pres.; Dr. O. W. Harris, Jr., Liberty, Secy.

Eleventh District Society, Crockett, Spring, 1954. Dr. John Dean, Crockett, Pres.; Dr. Marlin T. Braswell, Henderson, Secy.

Twelfth District Society, Waco, January, 1954. Dr. Howard Dudgeon, Jr., Waco, Pres.; Dr. Walter B. King, Jr., Waco, Secy.

Thirteenth District Society, Fort Worth, 1954. Dr. William E. Crump, Wichita Falls, Pres.; Dr. Robert D. Moreton, 815 Medical Arts Bldg., Fort Worth, Secy.

Fourteenth District Society, Denton, 1954. Dr. J. W. Atchison, Gainesville, Pres.; Dr. L. W. Johnston, 502 W. College St., Terrell, Secy.

Fifteenth District Society, Longview, April 20, 1954. Dr. L. E. Rutledge, Daingerfield, Pres.; Dr. George Tate, Longview, Secy.

CLINICS

Dallas Southern Clinical Society, Dallas, March 15-18, 1954. Miss Helga Boyd, Medical Arts Bldg., Dallas 1, Executive Secy.

Central Texas Spring Clinic, Waco, April 7, 1954. Dr. Walter B. King, 2320 Columbus Ave., Waco, Secy.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 26-28, 1954. Dr. John M. Smith, Jr., 205 Camden St., San Antonio, Secy.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1954. Dr. Maurice E. St. Martin, Room 103, 1430 Tulane Ave., New Orleans 12, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference. Dr. C. H. Wilson, Wichita Falls, Chairman.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 25-28, 1954. Miss Alma F. O'Donnell, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Postgraduate Medical Assembly of South Texas, July 19-21, 1954. Dr. Edward T. Smith, 906 Hermann Professional Bldg., Houston, Secy.

State Tumor Conference, Wichita Falls, March 31, 1954. Dr. Bailey R. Collins, 925½ Scott Street, Wichita Falls, Director.

BOARD EXAMINATIONS

Texas State Board of Examiners in Basic Sciences. Mrs. Betty Ratcliff, 407 Perry-Brooks Bldg., Austin, Chief Clerk.

Texas State Board of Medical Examiners, Fort Worth, June 21-23, 1954. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Secy.

Postgraduate School of Medicine Courses

A number of courses will be offered by the various branches of the University of Texas Postgraduate School of Medicine beginning in January. They are as follows:

Gastroenterology—Tuesday evenings January 12-April 20, Houston. Instructor in charge: Dr. Edward C. Malewitz, Houston. Topics: common medical diseases of the mouth, benign esophageal lesions, surgery of esophagus and diaphragmatic hernia. Tuition fee: $25. Credit hours: 30.

Pediatrics—Monday evenings, January 11-March 1, Austin. Instructor in charge: Dr. David Schrum. Topics: management of allergic child, allergic problems as seen in general practice, differential diagnosis between mongolism and cretinism, and care of the sick child. Tuition fee: $25. Credit hours: 16.

Office Traumatic Surgery and Orthopedics—Wednesday evenings, January 27-March 17, Corpus Christi. Tuition fee: $25. Credit hours: 16.

Medicine—January 15-16, Galveston.

Systemic Mycoses—January 18-20, Dallas.

Courses for the remainder of the year are scheduled to begin on the following dates:

Houston: Wet clinics in gynecology, February 2; ocular motor anomalies, February 1; heart seminar, February 15; cancer symposium on fundamental cancer research, early April.

Austin: dermatology, March 8.

San Antonio: clinical psychiatry, February 3.

Temple: medical and surgical conferences, March 1.

Galveston: surgery, February 12; obstetrics, April 16.

Dallas: orthopedic surgery, February 15; surgery, April 19.

Further information about any of the courses listed may be obtained by writing the following persons: Houston, Austin, Corpus Christi and Temple courses: Dr. Mavis P. Kelsey, Acting Dean, University of Texas Postgraduate School of Medicine, Texas Medical Center, 1400 Outer Belt Drive, Houston 25.

Galveston courses: Dr. E. Ivan Bruce, Jr., Director, Postgraduate Division, University of Texas Medical Branch, Galveston.

Dallas courses: Dr. John S. Chapman, Assistant Dean for Graduate and Postgraduate Education, Southwestern Medical School of University of Texas, 2211 Oak Lawn Avenue, Dallas.

PERSONALS

Dr. Homer E. Prince, Houston, is a trustee of the newly established American Foundation for Allergic Diseases, sponsored jointly by the American Academy of Allergy and the American College of Allergists. The foundation, with headquarters in New York, expects to promote education in the problems of allergy among members of the medical profession and the public, to cooperate with medical institutions in developing facilities for the prevention and treatment of allergic diseases, and to provide facilities for research.

Dr. William C. Levin, Galveston, recently was named to the executive board of the American Association of Blood Banks.

Dr. Milford O. Rouse, Dallas, was elected to the executive committee of the Southern Medical Association at its convention in Atlanta, Ga., October 28.

Dr. T. M. Collins, Big Spring, who retired several years ago after practicing medicine fifty-five years, celebrated his eighty-fifth birthday at a barbecue dinner given by his sons September 2.

Dr. J. A. Allison, Grapevine, followed his regular routine of medical practice on his eighty-first birthday September 21, but more than seventy well-wishers visited his office.

Dr. and Mrs. J. Bartow Talley, Temple, are parents of a son, William Murphy, born October 14.

Dr. and Mrs. Joe G. Rape, Arlington, are parents of a boy born August 18, and *Dr. and Mrs. Percy Cook,* Mansfield, and *Dr. and Mrs. Arvel R. Ponton,* Fort Worth, are parents of girls born in October.

Dr. Charles H. Willingham, Whitehouse, father of *Dr. C. E. Willingham* of Tyler, councilor of the Eleventh District, died in September.

UNIVERSITY OF TEXAS MEDICAL BRANCH

Recent visitors to the University of Texas Medical Branch, Galveston, include Dr. Knud O. Moller, director of the Pharmacology Institute of the University of Copenhagen, whose subject for a seminar was treatment of barbital and morphine poisoning; Dr. Fritz Eichholtz, director of the Pharmacology Institute of the University of Heidelberg, who lectured at a special seminar on newly discovered effects of carbon dioxide; Dr. Francis O. Schmitt, chairman of the Department of Biophysics at Massachusetts Institute of Technology, whose lecture topic was electron microscope studies of connective tissue; Dr. Jack Caddum, of the Department of Pharmacology of the University of Edinburgh, who spoke on the design of experiments; and Dr. Walter Meek, of the Department of Physiology of the University of Wisconsin, who lectured on some physiologic effects of cardiac catheterization.

Additional visitors include General John Rees of London, director of the World Federation for Mental Health; Dr. Hans Selye, director of the Institute of Experimental Medicine and Surgery at the University of Montreal, who was presented by Phi Delta Epsilon fraternity in the Meyer Bodansky Memorial Lecture; Dr. Hugh de Wardener of St. Thomas' Hospital, London; Dr. Henry Viets, Boston; J. F. Toughs, professor of plastic surgery at the University of Glasgow; Sir Geoffrey Jefferson, professor of neurosurgery at the University of Manchester, England, who delivered the annual James Greenwood Lecture; Dr. Burton Baker, professor of anatomy at the University of Michigan, Ann Arbor; and Dr. David Green, director of the Laboratory on Enzyme Research at the University of Wisconsin.

Radio station KGBC of Galveston is featuring a weekly series of programs describing the activities of the University of Texas Medical Branch. General services, research, and teaching are being considered.

The Texas Academy of Science met at the Medical Branch December 4-6.

A gift of $12,000 to build special quarters in the Ashbel Smith Building to serve as a student lounge has been contributed anonymously. The new facility will contain student, committee, and meeting rooms as well as a general lounge for recreation and social gathering.

Dr. Kenneth N. Earle of the Neurology Institute at McGill University has been appointed associate professor of neuropathology at the Medical Branch.

Chauncey D. Leake, Ph. D., vice-president of the University of Texas School of Medicine, presented several addresses at the world conference on medical education held in London in August.

Dr. George R. Herrmann, director of Latin-American relations at the Medical Branch, was one of the speakers at a seminar on cardiovascular conditions at the University of Puebla, Mexico, in October.

Southwestern Medical School

A course in systemic mycoses, open to any interested physician up to a maximum registration of 100, will be offered January 18-20 by Southwestern Medical School of the University of Texas, Dallas. Guest speakers will include Drs. Libero Ajello, Mycology Laboratory, United States Public Health Service, Chamblee, Ga.; Norman Conant, professor of mycology, Duke University School of Medicine, Durham, N. C.; M. L. Furcolow, senior surgeon, United States Public Health Service, University of Kansas Medical Center, Kansas City; and C. E. Smith, dean of the School of Public Health, University of California, San Francisco. Fee for the course will be $35. Advance registration will be accepted by Dr. John S. Chapman, assistant dean of graduate and postgraduate education at Southwestern.

A postgraduate course in obstetrics and gynecology was offered by the Southwestern Medical School of the University of Texas, Dallas, December 7-9, especially for general practitioners. Full-time and clinical staff members of the school touched on infection, hemorrhage and toxemia, primary menstrual disturbances, carcinoma, hysterectomy, cervicitis, prenatal care, and operative obstetrics.

Dr. William F. Mengert, professor of obstetrics and gyne-

cology at Southwestern Medical School, has been named chairman of the obstetrics and gynecology test committee of the National Board of Medical Examiners.

Dr. George N. Aagaard, dean of Southwestern, has been elected to a three-year term on the executive council of the Association of American Medical Colleges.

Dr. Leonard A. Scheele, surgeon general of the United States Public Health Service, Washington, spoke to members of the Southwestern faculty when he was in Dallas early in September.

San Antonio Assembly Names Speakers

Speakers for the eighteenth International Medical Assembly of Southwest Texas, scheduled for January 26-28 at San Antonio, have been announced as follows:

DR. HIRAM W. K. BATSON, Tulane University of Louisiana School of Medicine, New Orleans.

DR. CONRAD BERENS, Columbia University College of Physicians and Surgeons, New York.

DR. BAYARD CARTER, Duke University School of Medicine, Durham, N. C.

DR. HAROLD W. DARGEON, Memorial Hospital, New York.

DR. AUSTIN I. DODSON, Medical College of Virginia, Hospital Division, Richmond.

DR. FRANKLIN G. EBAUGH, University of Colorado School of Medicine, Denver.

DR. THOMAS P. FINDLEY, JR., Ochsner Clinic, New Orleans.

DR. ROBERT B. KERR, University of British Columbia, Vancouver.

DR. FRANCIS L. LEDERER, University of Illinois College of Medicine, Chicago.

DR. HAYES MARTIN, Memorial Hospital, New York.

DR. ALAN R. MORITZ, Western Reserve University School of Medicine, Cleveland.

DR. JAMES T. PRIESTLEY, Mayo Clinic, Rochester, Minn.

DR. OVAR SWENSON, Harvard University Medical School, Boston.

DR. C. H. WATKINS, Mayo Clinic, Rochester, Minn.

DR. A. JUSTIN WILLIAMS, University of California Medical School, San Francisco.

DR. CARROLL S. WRIGHT, Temple University School of Medicine, Philadelphia.

Special features of the three-day assembly include "Ask the Expert" panels on two surgical and two medical topics: "The Acute Abdomen," "Differential Diagnosis of Peptic Ulcer and Carcinoma," "Disturbed Nonmalignant Bowel Function," and "ACTH and Cortisone." Also, medical and surgical clinicopathologic conferences will be held, as will a symposium on psychosomatic medicine. Guest speakers for the symposium will include a psychiatrist, an internist, and a dermatologist.

There will be a dinner dance January 26 and a stag party January 27. Entertainment for visiting wives is being planned as well. The registration fee of $20 will cover all aspects of the meeting.

Details of the assembly program, open to physicians affiliated with their county medical societies, may be secured from Dr. John M. Smith, Jr., 205 Camden, San Antonio, secretary.

HELP IN BLOOD PRESSURE STUDY ASKED

The cooperation of physicians throughout the nation has been requested by Dr. Arthur M. Master, Herbert H. Marks, and Dr. Harry L. Jaffe, all of New York, who are undertaking a statistical study of blood pressure in persons past 65 years of age. Questionnaires asking for readings of pressures in 6 persons have been sent to 17,000 physicians. The American Medical Association has given aid to the investigation, which is sponsored by the New York Heart Association, Mount Sinai Hospital of New York, and the Metropolitan Life Insurance Company. The current study is an extension of the survey on 74,000 gainfully employed persons up to age 64, the results of which were published in 1950.

ARTHRITIS MORTALITY FIGURES

Analysis of a series of 583 patients with rheumatoid arthritis treated over a twenty year period at Massachusetts General Hospital, in which 137 deaths occurred, revealed that infections, particularly pneumonia and septicemia, were responsible for 25 per cent of deaths, a higher percentage than expected in the normal population.

According to a communication from Sidney Cobb, a member of the faculty at the University of Pittsburgh Graduate School of Public Health, cited in "Spectrum" in the October 17 *Journal of the American Medical Association,* none of the 137 deaths occurred as a direct result of arthritis, but patients who contracted the disease prior to 25 years of age experienced a loss of eight years from the usual life expectancy; those who became ill between 25 and 49 years of age lost two or three years from the life expectancy; and those in whom rheumatoid arthritis began after they were 50 years of age experienced no appreciable change in life expectancy.

PAN AMERICAN SESSIONS SET

More than 2,500 doctors and medical researchers from twenty-two nations of the Western Hemisphere will exchange information during a sixteen-day congress. The Pan American Medical Association will meet daily after embarkation from New York on January 6 and will participate in specially planned activities in Caracas, San Juan, Ciudad Trujillo, St. Thomas, and Havana. Dr. Charles Crocker, Pebble Beach, Calif., is executive secretary of the organization.

A second medical adventure in Latin America awaits members of the Pan American Academy of General Practice, who will convene in Medellin, Colombia, February 15-20 and then visit Bogota, Barranquilla, and Montega Bay, Jamaica. Medicine, surgery, and pharmacy will be considered on the program. Registration for the congress and for transportation via plane or steamship may be made through Dr. Arturo Martinez, secretary, 500 West End Avenue, New York.

INTERNATIONAL SURGEONS TO CONVENE

The Southeastern District meeting of the International College of Surgeons, open to interested physicians and surgeons of Texas, will be held January 29-30 in West Palm Beach, Fla. The reservation fee of $10 will cover an informal dinner, and special events will be arranged for visiting ladies. Reservations should be made by writing Dr. Lloyd J. Netto, 416 Comeau Building, West Palm Beach, Fla., or the Pennsylvania Hotel, West Palm Beach.

Cancer Research Grant to Anderson Hospital

The M. D. Anderson Hospital for Cancer Research, Houston, is recipient of a $75,000 research grant from the American Cancer Society for use during the current year. This is the seventh consecutive annual allocation from the society to the hospital, which in all has received $340,776.52 from this source. American Cancer Society research funds now at work in Texas total $140,595.

World Medical Association

The World Medical Association will hold its 1954 meeting in Rome, September 26-October 2. Members of the American Medical Association are invited to attend. The World Medical Association, 345 East 46th Street, New York 17, will secure transportation, hotel reservations, and assist in planning side tours of Europe.

LIBRARY SECTION

CESAREAN SECTION

The cesarean section is one of the paradoxes of medicine, and until recently, one of the tragedies. For many years it was thought the name of cesarean section originated with the birth of Julius Caesar, who was brought into the world by a cesa matris uteri. Since Julia, his mother, lived many years after his birth and such operations were not then performed on living women, this theory has been proved incorrect.

Another theory that is possible but not given much credence is that it originated from the Latin word *caedere* (to cut). At any rate, in 715 B. C. Numa Pompilius, the King of Rome, commanded that all women who died late in pregnancy have an abdominal section performed, first, to save the child if possible and, second, to allow a separate burial for mother and child in the event both died.

From the earliest records of the cesarean section until 1500 there were no indications to suggest that this operation was performed on a living woman. From 1500 to 1876 the operation was performed occasionally when all other methods failed. The first cesarean section as it is generally accepted today was performed by Jeremias Trautman in Wittenberg, Germany, in 1610 on a woman with hernia uteri. In the United States it is thought that Dr. Jessee Bennett of Rockingham, Va., on January 14, 1794, performed the operation on his wife, saving both the mother and the child.

There have been many controversies over who did and who did not perform the operation as it was not generally acceptable because of its high mortality rate. It was many years before it was established that Dr. Francois Marie Prevost performed the first successful cesarean section in Louisiana and in the Deep South. Dr. Elias Samuel Cooper, founder of the first medical school in California, performed a cesarean section and was later sued for $25,000.

And thus it goes through medical history. Kayser of Copenhagen in 1844 reported a 62 per cent mortality from cesarean sections; L. F. McLean in 1949 reported 0.3 per cent mortality out of 339 sections.

The following articles and motion pictures in this general field may be of interest:

ARTICLES

McLean, L. F., and others: Present-Day Safety of Cesarean Section, Am. J. Obst. & Gynec. *60*:860-65 (Oct.) 1950.

Vogt, W. H.: Cesarean Section, J. Okla. M. A. *25*:26-33 (Jan.) 1932.

FILMS

Extraperitoneal Cesarean Section (describing a simplified paravesical technique), 16 mm., sound, color, 23 minutes. (Purchased by Texas Medical Association Memorial Library.)

CONTRIBUTIONS TO LIBRARY

Grateful acknowledgment is made by the Texas Medical Association Memorial Library for the following recent gifts:

Dr. John Connolly, Shiner: 2 books.

Dr. and Mrs. Ramsay Moore, Dallas: 4 books, donated through the Woman's Auxiliary to the Texas Medical Association.

Dr. and Mrs. M. B. Porter, Austin: 3 books, 2 journals, 1 pamphlet.

Dr. M. B. Porter, Austin: 2 books.

Dr. R. L. Powers, San Angelo: 1 16 mm. motion picture.

Dr. N. L. Schiller, Austin: 16 journals, 10 reprints.

BOOKS RECEIVED IN NOVEMBER

Bische, Louis E.: Cure Your Nerves Yourself, New York, Wilfred Funk, Inc., 1953.

Ciba Foundation: Visceral Circulation, Boston, Little, Brown and Company, 1953.

Engle, F. L.: Pregnancy Wastage, Springfield, Ill., Charles C Thomas, 1953.

Haymaker, Webb, and Woodhall, Barnes: Peripheral Nerve Injuries, Principles of Diagnosis, Philadelphia, W. B. Saunders Company, 1953.

Klendshoj, Niels C.: Fundamentals of Biochemistry in Clinical Medicine, Springfield, Ill., Charles C Thomas, 1953.

Koch, Frederick C., and Hanke, Martin E.: Practical Methods in Biochemistry, Baltimore, Williams and Wilkins Company, 1953.

Means, J. H.: The Thyroid and Its Diseases, ed. 2, Philadelphia, J. B. Lippincott Company, 1948.

Moore, Daniel C.: Regional Block, Springfield, Ill., Charles C Thomas, 1953.

Noble, Frank W.: Electrical Methods of Blood-Pressure Recording, Springfield, Ill., Charles C Thomas, 1953.

Page, I. H.: Hypertensive Disorders of Pregnancy, Springfield, Ill., Charles C Thomas, 1953.

Parsons, Langdon, and Ulfelder, Howard: An Atlas of Pelvic Operations, Philadelphia, W. B. Saunders Company, 1953.

Seed, Lindon, and Fields, Theodore: Treatment of Toxic Goiter with Radioactive Iodine, Springfield, Ill., Charles C Thomas, 1953.

Smul, Josef S.: Respiratory Diseases and Allergy, New York, Medical Library Company, 1953.

Spurling, R. Glen: Lesions of the Lumbar Intervertebral Disc, Springfield, Ill., Charles C Thomas, 1953.

U. S. Department of Health, Education, and Welfare: Mental Health Implications in Civilian Emergencies, 1953.

Weil, Alfred J., and Saphra, Ivan: Salmonellae and Shigellae, Laboratory Diagnosis Correlated with Clinical Manifestations and Epidemiology, Springfield, Ill., Charles C Thomas, 1953.

LIBRARY REQUESTS INCREASE

The Library staff is gratified with the response to literature on use of Library services mailed to members of the Texas Medical Association in October. More than 400 requests for material were received in three days. To search the literature for so many requests takes time, but the staff hopes no one was inconvenienced with any delay in receiving the material requested. It is hoped that this usage of the Library will continue, and every effort will be made to answer each request within twelve hours if possible.

MOTION PICTURES FOR LOAN

Office Courtesy

16 mm., sound, color, 12 minutes. (Purchased by Texas Medical Association Memorial Library.)

This timely film in human relations dramatizes techniques in meeting and working with people, face to face and over the telephone. It portrays a secretary who did not know how to meet the public. Her disagreeable manner drove people away. Then came a change of attitude, and the same secretary is shown in the same situations as they should be handled. This film is excellent for secretaries, office nurses, and nursing training classes.

Extraperitoneal Cesarean Section

16 mm., sound, color, 17 minutes. (Purchased by Texas Medical Association Memorial Library.)

Prepared by Dr. A. R. Abarbanel, this film demonstrates the relative ease and safety with which extraperitoneal cesarean section may be performed. It stresses the avoidance of trauma to the bladder and of entrance into the peritoneal cavity. The film is well organized and the photography and narration are excellent. It should be of interest to county medical society groups.

Hypodermic Syringes and Needles: Their Care and Function

16 mm., sound, color, 62 minutes. (Courtesy of Becton, Dickinson and Company.)

This film shows the basic standards for syringes and needles and presents the accepted techniques for their care. Proper use and simple injection techniques are demonstrated. This is an excellent film for nurses and nursing trainees and will be of interest to anyone concerned with training schools.

Surgical Treatment for Splenic Flexure Carcinoma with Solitary Liver Metastasis

16 mm., silent, color, 40 minutes. (Courtesy of Dr. Philip Thorek, Chicago.)

The patient in this film has a large bowel obstruction from the splenic flexure carcinoma. "Blind cecostomy" is performed to relieve obstruction. Two weeks later partial colectomy with an aseptic end-to-end anastomosis is done, together with partial left lobe hepatectomy for a large solitary metastatic nodule. Closure of the cecostomy is demonstrated. Illustrations are included to amplify the procedures. The photography is excellent.

BOOK NOTICES

[1]Familial Nonreaginic Food Allergy

ARTHUR F. COCA, M. D., *Oradell, N. J.; with contributions by* CONRAD BERENS, M. D., *New York;* EDITH L. CUMMING, *Valley Cottage, N. Y.;* LOUIS J. GIRARD, M. D., *New York;* LEONARD S. GREEN, L.L.B., *New York;* ALAN JOHNSTON, M. D., *Plainfield, Ind.;* ARTHUR P. LOCKE, Ph. D., *New Brunswick;* MILO G. MEYER, M. D., *Michigan City, Ind.;* M. MURRAY PESHKIN, M. D., *New York; and* SUMNER PRICE, M. D., *Honolulu, T. H. Third edition. 279 pages. $10.50. Springfield, Ill., Charles C Thomas, 1953.*

The book elaborates the author's theory of so-called familial nonreaginic food allergy. He states that it affects 90 per cent of the population and differentiates it from the usually recognized atopic diseases which affect only from 7 to 10 per cent of people. The coined term "idioblapsis," of Greek origin, alludes to a supposed individual peculiarity or sensitivity to food; literally it means "one's own destroyer." Almost any organ may be the shock tissue and the pathologic process consists of edema under pressure or ulceration. Thus, he accounts for just about any symptom that can be imagined.

Listed as examples of the ubiquitous syndrome are headache, all functional gastrointestinal complaints, canker sores, peptic ulcer, constipation, hemorrhoids, fatigue, fearfulness, hives, multiple sclerosis, dysmenorrhea, colds, hypertension, eczema, arthritis, neuralgia, glaucoma, and many other manifestations. Poliomyelitis, epilepsy, and psychoses are admitted into the outer portals but are not given full membership in this far from exclusive group.

The method for diagnosing the offending foods and thus paving the way for cure of the malady lies in the "pulse-diet" record, according to the author. If the daily pulse range exceeds 20 (later stated to be 16) and if the daily maximal counts per minute vary more than two beats, or if the maximum is 88 or more, it proves that a person is the victim of nonreaginic food allergy. To spot the offending agent single foods are eaten five times a day. Three counts are made at thirty minute intervals after eating. If the pulse rate increases more than the normal range, it is proof of sensitivity.

The author states that no case of infantile eczema has failed to respond to his method of treatment. For the few cases of other disorders that failed to respond, usually because patients were thought to be sensitive to too many foods, he advocates surgical sympathectomy.

Completely inadequate proof is submitted for most of the assertions made in the book. If there is any merit in the author's method, in the ten years since the first edition was published he should have been able to document a large series of cases of almost any type. The ones cited are meager and not at all convincing. The author feels that allergists as well as other physicians do not take his work seriously, and on that one score he is certainly correct.

[2]Mid-Century Psychiatry

Edited by ROY R. GRINKER, M. D., *Director of Institute for Psychosomatic and Psychiatric Research and Training, Michael Reese Hospital, Chicago. 183 pages. $5.50. Springfield, Ill., Charles C Thomas, 1953.*

This book is a carefully integrated symposium of present-day psychiatry in which thirteen prominent contributors blend their "multidisciplinary" ideas toward a united understanding of human behavior. The reader will find "patchworks of concepts loosely basted together . . . which do not satisfactorily achieve multilevel correlations. . . ." The collaborators attempt to consolidate the gains of biological, psychological, and social understandings of human behavior in our times.

The book is a "must" for the student of psychiatry, and it should add to his enthusiasm.

Human Locomotion and Body Form

DUDLEY J. MORTON, M. D., *Formerly Associate Professor, Department of Anatomy, also Associate Clinical Professor, College of Physicians and Surgeons, Columbia University; with the collaboration of* DUDLEY DEAN FULLER, *Associate Professor, Department of Mechanical Engineering, Columbia University. 285 pages. $5. Baltimore, Williams and Wilkins Company, 1952.*

This text on the anatomy and physiology of the foot is divided into two parts. Part One has to do with the underlying principles of adaptation in which the author discusses gravity, the foot in walking, and locomotor habits. Part Two is an analysis of the walking stride, discussing metabolism and locomotion, histologic and chemical aspects, human mentality, and bipedism.

[3]Essentials of Infant Feeding for Physicians

HERMAN FREDERIC MEYER, M. D., *Assistant Professor, Department of Pediatrics, Northwestern University School of Medicine; Associate Attending Physician, Children's Memorial Hospital, Chicago. 252 pages. $6.75. Springfield, Ill., Charles C Thomas, 1952.*

This book is a concise, well written monograph concerning the feeding of infants. Dr. Meyer has approached the

[1]*A. Ford Wolf, M. D., Temple.*

[2]*Lee F. Scarborough, M. D., Austin.*

[3]*George Clifford Thorne, M. D., Austin.*

subject from the viewpoint of a practicing pediatrician and has produced a useful book. His methods of infant feeding are practical and undoubtedly effective.

I recommend this book without hesitation.

[4]The Anatomy of the Nervous System

STEPHEN WALTER RANSON, M.D., Ph. D., *Late Professor of Neurology and Director of Neurological Institute, Northwestern University Medical School, Chicago. Revised by* SAM LILLARD CLARK, M. D., Ph. D., *Professor of Anatomy, Vanderbilt University School of Medicine, Nashville. Ninth edition. 581 pages. $8.50. Philadelphia, W. B. Saunders Company, 1953.*

The ninth edition of this familiar textbook of neuroanatomy has been revised thoroughly by Dr. Clark. The chapter on contents has been rearranged to include a more readily identifiable index form. A laboratory outline of neuroanatomy is included and will doubtless help the student to blend the account of structure with function, and the section on neurologic stains is particularly valuable. Dr. Clark has added twenty-six illustrations, some of which are in color, and has succeeded in bringing the text up to date in the field of functional aspects of the cerebral cortex.

There is an extensive bibliography and a full list of references to the literature. There is no detailed presentation of evidence concerning new or disputed points, but the authority for the point is always cited and the student may refer to literature for a fuller discussion of the subject. Dr. Clark lays stress on the presentation of neuroanatomy from the dynamic rather than the static point of view, and it is apparent that he has succeeded well in this ninth edition. The book remains one of the outstanding textbooks in the field of neuroanatomy and has been widely adopted.

[5]Current Therapy 1953

HOWARD F. CONN, M. D., *Editor; Consulting Editors:* M. EDWARD DAVIS, VINCENT J. DERBES, GARFIELD G. DUNCAN, HUGH J. JEWETT, WILLIAM J. KERR, PERRIN H. LONG, H. HOUSTON MERRITT, PAUL A. O'LEARY, WALTER L. PALMER, HOBART A. REIMANN, CYRUS C. STURGIS, *and* ROBERT H. WILLIAMS. *835 pages. $11. Philadelphia, W. B. Saunders Company, 1953.*

Here is a volume dedicated strictly to current treatment, described in concise manner and dealing with almost every known disease encountered by a physician in general practice. It probably will prove valuable to specialists, too, as a handy reference but in a more limited way.

The text is divided into sixteen sections, each dealing with the separate systems of the body. Each section has been reviewed by a consultant in the specialized field. Various contributors, many of whom are well known, wrote the articles. To pick out the best section in the book would be difficult. Needless to say, the sections on tuberculosis, rheumatic fever, allergy, and obstetrics are especially informative.

The book is printed in clearly legible style which makes for easy reading with a minimum of searching for specific details. The index is thorough and any subject is quickly found. A roster of drugs, including packaging data and manufacturer, is added.

The use of cortisone in acute rheumatic fever is discussed, pro and con. The general opinion is that the hormone shortens the course of the disease and prevents or reduces primary or recurrent cardiovascular damage. The long range effect of cortisone, however, is not known. On the other hand, neither salicylates nor hormones are the full answer to rheumatic fever at present.

An interesting new fact, noted in the treatment of snake bite, is that incision and drainage with suction is no longer advocated. The therapy now centers around antivenins and antibiotics for secondary infection.

Summarizing, this text seems to be in keeping with the best traditions of modern medicine and is well worth its price in knowledge gained.

[6]Handbook of Orthopaedic Surgery

ALFRED RIVES SHANDS, JR., M. D., *Medical Director of the Alfred I. duPont Institute of the Nemours Foundation, Wilmington, Del.; Visiting Professor of Orthopedic Surgery, University of Pennsylvania School of Medicine, Philadelphia. In Collaboration with* RICHARD BEVERLY RANEY, M. D., *Professor of Surgery in Orthopedic Surgery, University of North Carolina, Chapel Hill. Illustrated by* JACK BONACKER WILSON *and others. Fourth edition. 644 pages. $8. St. Louis, C. V. Mosby, 1952.*

On the whole this book is excellent for teaching purposes both for the medical student and for the general practitioner, but it will have to be supplemented by other texts since no mention is made of fresh fractures.

The book is divided into twenty-four chapters, each of which had been reviewed before publication by a recognized authority. There are a few things omitted; for example, no mention is made of gamma globulin in chapters on poliomyelitis; in the chapter on arthritis no mention is made of postpartum plasma in atrophic arthritis; and in the chapter on tuberculosis of bones and joints no mention of isonicotinic acid therapy is made.

The last half of the book is divided into chapters on various body regions. Affections involving each region are discussed together with differential diagnoses and treatment. This is an excellent arrangement for teaching purposes.

[7]Bacterial and Mycotic Infectons of Man

Edited by RENE J. DUBOS, PH. D., *The Rockefeller Institute for Medical Research. 886 pages. $7.50. Philadelphia, J. B. Lippincott, 1952.*

This book is devoted to the exposition of the principles that govern the growth and development of pathogenic bacteria, actinomycetes, and molds and the reactions of the host to their presence. Viruses and Rickettsia are discussed in a companion volume edited by Rivers. This book was not intended as a laboratory manual; therefore, details of techniques, media, and isolation are omitted for the most part. There are chapters on the general morphology, physiology, and immunology of bacteria as well as extensive discussions of individual species. Sections on the principles of action and the uses of the antibiotic drugs and paragraphs on the chemotherapy of individual organisms will interest physicians. They are unusually up to date for a textbook. There is a brief but useful chapter on applied medical bacteriology in which the normal and pathologic flora of anatomic regions is presented as an aid to the diagnostician. A review of human blood groups by Levine is a valuable if somewhat ectopic inclusion.

The book is well planned and extremely well edited. Uniformity of chapter structure and consistently excellent writing have been maintained despite the large number of contributors. Enough space was allotted to permit full discussions of important subjects, but the text never becomes involved or tedious. The material is printed on good paper in the popular double columns. Illustrations are neither numerous nor of the best quality, except in the section on mycosis. This is not a major defect for the average reader and helps

[4]*James R. Blair, Jr., M. D., San Antonio.*
[5]*H. C. Powell, M. D., Austin.*
[6]*K. J. Kemp, M. D., Corpus Christi.*
[7]*Max Rottenstein, M. D., Laredo.*

keep the price low. The references are complete and current. They are so presented that they do not clutter the discussions. The volume is recommended to all who want detailed, timely information on mycology and bacteriology.

[8]The Principles and Practice of Medicine

L. S. P. DAVIDSON, M. D., F.R.C.P.(Edin.) F.R.C.P (Lond.), *Physician to Her Majesty the Queen in Scotland; Professor of Medicine and Clinical Medicine, University of Edinburgh. 919 pages. $6.75. Edinburgh, E. & S. Livingstone, Ltd., 1952. Distributed by Williams and Wilkins Company, Baltimore.*

This is a good book on the subject of general medicine. The author has presented the material in a manner which is easily read. He does not go into any great detail but is concise and to the point.

The author discusses the diseases of each system of the body by chapters. In each chapter he discusses the diseases under the headings of etiology, clinical features, pathologic features, complications, diagnosis, prophylaxis, and treatment. Also is included a discussion of the newer antibiotics and their uses.

[9]Malignant Disease and Its Treatment by Radium

SIR STANFORD CADE, F.R.C.S., M.R.C.P., F.F.R. (Hon.), *Surgeon, Westminster Hospital; Consulting Surgeon, Mount Vernon Hospital and Radium Institute. With a foreword by* SIR ERNEST ROCK CARLING, F.R.C.P., F.R.C.S., F.F.R., *Consulting Surgeon and Vice-President Westminster Hospital. Volume four. 544 pages. $12.50. Baltimore, Williams and Wilkins Company, 1952.*

The object of this book is to assist the radiotherapist in his task, to help the surgeon in the selection of treatment, and to provide all doctors with an account of the clinical and pathologic aspects of malignant disease and of the means of treatment available today.

The series of volumes is divided into three parts. The first deals with the physics of radiation. The biologic aspects of radiation are described in the second part. The third and main part of the book, which describes the natural history and treatment of malignant disease, is divided into twenty chapters dealing with various anatomic sites, each in turn giving the natural history of the disease, the choice of method of treatment, and the technique of radiation therapy. Volume four contains the last eight chapters of this section. It deals with a miscellaneous group of tumors and has been almost entirely rewritten.

Successful treatment of malignant disease depends upon three main factors: a sound knowledge of the disease; a wise selection of the method of treatment; and an accurate and skillful technique in administering this treatment. The chapters in this volume are detailed, are interesting, and definitely give a precise and complete study of malignant disease in all phases.

[10]Poliomyelitis

W. RITCHIE RUSSELL, M. D. (Edin.), F.R.C.P. (Lond.), *Consultant Neurologist to the United Oxford Hospitals; Clinical Lecturer in Neurology, University of Oxford. 84 pages. $3. London, Edward Arnold and Company, 1952. Distributed by Williams and Wilkins Company, Baltimore.*

This monograph is succinctly written and highly readable. It is replete with concise case reports, and the author demonstrates the role of stress effectively. The author's use of sedation in the bulbar cases and the use of analgesics in early cases will be viewed with considerable misgivings in this area. The author is more conservative in the use of tracheotomy than the present trend in this country, but he does advocate very early passive motion and has devised methods for evaluating muscle strength.

[11]Essentials of Medical Diagnosis

RT. HON. LORD HORDER, G.C.V.O., M. D., F.R.C.P. *(Lond.), Extra Physician to H. M. the Queen; Consulting Physician to St. Bartholomew's Hospital; and* A. E. GOW, M. D., F.R.C.P. *(Lond.), Honorary Physician to Household, H. R. H. the Duchess of Kent; Consulting Physician, St. Bartholomew's Hospital. Revised with the assistance of* RONALD BODLEY SCOTT, M. A., D. M. *(Oxon),* F.R.C.P. *(Lond.), Physician to H. M. Household; Assistant Physician and Senior Demonstrator of Practical Medicine, St. Bartholomew's Hospital. Second edition. 462 pages. $6. Baltimore, Williams and Wilkins Company, 1953.*

The second edition of this work comes with many revisions which bring the interpretation of diagnostic material into line with present-day concepts.

It is not the usual book of physical diagnosis; however, the primary emphasis is there laid. The material is covered in regional distribution with a short but concise section on the anatomy and physiology of the system being investigated, followed by the description of proper methods of examination with a rather full interpretation of pathologic findings. Although the emphasis is on physical diagnosis, there is a concise interpretation of abnormal laboratory and roentgenographic findings to be expected.

The wealth of information contained in the short span of this work is extremely well presented and arranged in such a manner that the interest and attention of the reader remains stimulated throughout.

In this book the authors have developed a logical, useful, and interesting method of approach to the problems of medical diagnosis which should prove useful to both the student and the practitioner of medicine.

[12]Gynecology: Diseases and Minor Surgery

Compiled and Edited by ROBERT J. LOWRIE, M. D., F.A.C.S., D-OG., *Associate Clinical Professor of Obstetrics and Gynecology, College of Medicine, New York University; Attending Gynecologist and Obstetrician, St. Vincent's Hospital. 806 pages. $22.50. Springfield, Ill., Charles C Thomas, 1952.*

It is a great pleasure to read a volume as simply and carefully compiled as this book. The author has managed beautifully to get together the basic and more intricate phases of gynecology from contributions of accepted leaders in the field and put them together with painstaking care, therefore making not only a contribution to gynecologic literature but an artistic contribution as well.

There are seventy-eight contributors to this volume, and the book is divided not only into sections, chapters, indexes, and bibliography, but each chapter has its own index and bibliography. Also, each subject in each chapter is outlined and divided, by the use of large type, into definition, incidence, etiology, pathology, signs and symptoms, differential diagnosis, and therapy. In each chapter the facts are portrayed, and there is no confusion from quoting too many opinions.

The use of italics is outstanding and helps considerably in gleaning the facts portrayed. Illustrations are voluminous, and the explanation concerning the illustrations is in good detail.

[8]*Rush A. Snyder, M. D., Canadian.*
[9]*L. C. Paggi, M. D., Austin.*
[10]*J. M. Coleman, M. D., Austin.*
[11]*William E. Jones, M. D., Texarkana.*
[12]*G. E. Peacock, M. D., Big Spring.*

This volume is highly recommended as a reference book for general practitioners and those physicians specializing in gynecology. It is also recommended as a textbook for junior and senior medical students as well as residents in gynecology. This volume would be an asset to any physician's library, no matter what his practice or interest.

[13]Textbook of Surgery

Edited by H. F. MOSELEY, D. M., F.A.C.S., F.R.C.S. (C), *Assistant Professor of Surgery, McGill University; Associate Surgeon, Royal Victoria Hospital, Montreal, Canada. With a foreword by* G. GAVIN MILLER, M. D., F.R.C.S. (C), F.A.C.S., *Chairman of Surgical Department, McGill University; Surgeon-in-Chief, Royal Victoria Hospital, Montreal. 896 pages. $15. St. Louis, C. V. Mosby Company, 1952.*

This book is well written. Its subject matter is well organized and well presented.

The illustrations, particularly above average in accuracy and clarity, are well spaced to cover the subject matter in an ample form; however, the book is exactly as described in its title, a textbook of surgery. An attempt is made to cover the entire field of surgery in 855 pages of text so that it may well be used for medical students as a textbook to introduce them to surgery, but it is entirely inadequate for the general practitioner or the surgeon or for reference by graduates in medicine.

In some instances an attempt is made to explain the physiology and pathology of the surgical disease, but this is too brief for any practical use.

This book is recommended only as a textbook for beginning students in surgery.

[14]Founders of Neurology

Prepared for the Fourth International Neurological Congress in Paris by Eighty-Four Authors, edited by WEBB HAYMAKER, M. D., *Chief, Neuropathology Section, Armed Forces Institute of Pathology, Washington, D. C., with bibliographic and editorial assistance by* KARL A. BAER, *Bibliographer, Army Medical Library, Washington, D. C. 479 pages. $10.50. Springfield, Ill., Charles C Thomas, 1953.*

This biographical sketch of 133 neuroanatomists, neuropsychiatrists, neuropathologists, clinical neurologists, and neurosurgeons is a masterpiece in itself and its references are complete. The perfectly condensed biographies from Achucarro, who delineated the nature of various cell types in the human cerebral cortex, to Clovis Vincent, the neurologic surgeon of France, give a clear description of the birthplace, forebears, education, and hospital and university appointments of these men. It also describes their association with each other and their teachers. It further depicts the work or works for which they are especially known in their respective neurologic fields, and last but not least gives the personal traits and characteristics, professional and social, of these interesting founders of neurology.

[15]Gynecologic and Obstetric Pathology

EMIL NOVAK, M. D., F.A.C.S., F.R.C.O.G. (Hon.), *Assistant Professor Emeritus of Gynecology, Johns Hopkins Medical School; Gynecologist, Bon Secours and St. Agnes Hospitals, Baltimore. Third edition. 595 pages. $10. Philadelphia, W. B. Saunders, 1952.*

In this, the third edition, the author combines his wealth of clinical experience as a gynecologist and obstetrician with pathologic laboratory findings in a more modern concept of pathology in compiling an excellent textbook. It is difficult to correlate this work with the general practice of gynecology and obstetrics. This text, however, was not intended for this purpose, as were some other works by the same author.

This work is written almost entirely from the teaching standpoint. And, while useful from the microscopic diagnosis standpoint, it is not so useful from a practitioner's standpoint.

Many of the common gross pathologic findings are not shown in illustrations and some are not even mentioned because of little serious importance. If these findings were more profusely illustrated in color and described with less emphasis on the microscope, the work would be more useful to the practitioner.

As a text it is excellent, but from a practical practicing standpoint it is not of much more help than older works. Its usefulness in fact is limited to the student and in the pathologic laboratory.

[16]Principles, Problems, and Practices of Anesthesia for Thoracic Surgery

HENRY K. BEECHER, M. D., *Henry Isaiah Dorr Professor of Research in Anesthesia, Harvard University; Director, Department of Anesthesia, Massachusetts General Hospital, Boston. Edited by* JOHN ADRIANI, M. D., *Director, Department of Anesthesia, Charity Hospital, New Orleans. A Monograph in American Lectures in Anesthesia. 65 pages. $2.50. Springfield, Ill., Charles C Thomas, 1952.*

This monograph is well written as is usual with Beecher. He writes to the point and in simple language that can be understood by surgeon and anesthetist alike.

Most of the book is taken up by the hazards encountered in lobectomy and pneumonectomy with a detailed explanation of methods and equipment necessary to overcome these hazards. These discussions cover respiratory obstruction, circulatory distress, anoxia, vagal reflexes, gas machines, endotracheal tubes, and controlled and assisted respiration.

This is followed by a discussion of pre-anesthetic medication, choice of anesthesia, and procedures after anesthesia is given.

There is a special section covering chest anesthesia for children, cardiac surgery, thoracoplasty, and miscellaneous chest operations.

The weak spot in this small book is Beecher's personal weakness. Being from Boston, he carries a running defense of ether throughout the entire book when in reality ether needs no defense. Anesthetists the world over recognize ether alone, mixed with nitrous oxide, or mixed with cyclopropane as an excellent agent for chest anesthesia.

The Ciba Collection of Medical Illustrations; Volume 1, Nervous System

A compilation of Pathologic and Anatomic Paintings prepared by FRANK H. NETTER, M.D., *with a foreword by* JOHN F. FULTON, M.D., *Sterling Professor of Medicine. 143 pages. $6. Commissioned and published by Ciba Pharmaceutical Products, Inc., Summit, N. J., 1953.*

This is the first of a series of Dr. Netter's drawings of the major anatomy and pathology of all the systems comprising the human organism. Dr. Netter is well known in the medical field for his excellent drawings. The many intricate illustrations are in color.

This book should be in every physician's library.

[13]*J. P. Lovett, M. D., Olney.*
[14]*C. E. Patterson, M. D., Dallas.*
[15]*William C. Holt, M. D., Angleton.*
[16]*Randle J. Brady, M. D., Houston.*

ORGANIZATION SECTION

Principles of Medical Ethics

of the

American Medical Association

Preamble

These principles are intended to serve the physician as a guide to ethical conduct as he strives to accomplish his prime purpose of serving the common good and improving the health of mankind. They provide a sound basis for solution of many of the problems which arise in his relationship with patients, with other physicians, and with the public. They are not immutable laws to govern the physician. The ethical practitioner needs no such laws; rather they are standards by which he may determine the propriety of his own conduct. Undoubtedly, interpretation of these principles by an appropriate authority will be required at times. As a rule, however, the physician who is capable, honest, decent, courteous, vigilant, and an observer of the Golden Rule, and who conducts his affairs in the light of his own conscientious interpretation of these principles, will find no difficulty in the discharge of his professional obligations.

Chapter I. General Principles

CHARACTER OF THE PHYSICIAN

SECTION 1.—The prime object of the medical profession is to render service to humanity; reward or financial gain is a subordinate consideration. Whoever chooses this profession assumes the obligation to conduct himself in accord with its ideals. A physician should be "an upright man, instructed in the art of healing." He must keep himself pure in character and be diligent and conscientious in caring for the sick. As was said by Hippocrates, "He should also be modest, sober, patient, prompt to do his whole duty without anxiety; pious without going so far as superstition, conducting himself with propriety in his profession and in all the actions of his life."

THE PHYSICIAN'S RESPONSIBILITY

SEC. 2.—"The profession of medicine, having for its end the common good of mankind, knows nothing of national enmities, of political strife, of sectarian dissensions. Disease and pain the sole conditions of its ministry, it is disquieted by no misgivings concerning the justice and honesty of its client's cause; but dispenses its peculiar benefits, without stint or scruple, to men of every country, and party and rank, and religion, and to men of no religion at all."*

GROUPS AND CLINICS

SEC. 3.—The ethical principles actuating and governing a group or clinic are exactly the same as those applicable to the individual. As a group or clinic is composed of individual physicians, each of whom, whether employer, employee or partner, is subject to the principles of ethics herein elaborated, the uniting into a business or professional organization does not relieve them either individually or as a group from the obligation they assume when entering the profession.

ADVERTISING

SEC. 4.—Solicitation of patients, directly or indirectly, by a physician, by groups of physicians or by institutions or organizations is unethical. This principle protects the public from the advertiser and salesman of medical care by establishing an easily discernible and generally recognized distinction between him and the ethical physician. Among unethical practices are included the not always obvious devices of furnishing or inspiring newspaper or magazine comments concerning cases in which the physician or group or institution has been, or is, concerned. Self laudations defy the traditions and lower the moral standard of the medical profession; they are an infraction of good taste and are disapproved.

EDUCATIONAL INFORMATION NOT ADVERTISING

SEC. 5.—Many people, literate and well educated, do not possess a special knowledge of medicine. Medical books and journals are not easily accessible or readily understandable.

The medical profession considers it ethical for a physician to meet the request of a component or constituent medical society to write, act or speak for general readers or audiences. The adaptability of medical material for presentation to the public may be perceived first by publishers, motion picture producers or radio officials. These may offer to the physician opportunity to release to the public some article, exhibit or drawing. Refusal to release the material may be considered a refusal to perform a public service, yet compliance may bring the charge of self seeking or solicitation. In such circumstances the physician should be guided by the decision of official agencies established through component and constituent medical organizations.

A physician who desires to know whether, ethically, he may engage in a project aimed at health education of the public should request the approval of the designated officer or committee of his county medical society.

The most worthy and effective advertisement possible, even for a young physician, especially among his brother physicians, is the establishment of a well merited reputation for professional ability and fidelity. This cannot be forced, but must be the outcome of character and conduct. The publication or circulation of simple professional cards is approved in some localities but is disapproved in others. Disregard of local customs and offenses against recognized ideals are unethical.

The promise of radical cures or boasting of cures or of extraordinary skill or success is unethical.

An institution may use means, approved by the medical profession in its own locality, to inform the public of its address and the special class, if any, of patients accommodated.

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*Sir Thomas Watson.

PATENTS, COMMISSIONS, REBATES AND SECRET REMEDIES

SEC. 6.—An ethical physician will not receive remuneration from patents on or the sale of surgical instruments, appliances and medicines, nor profit from a copyright on methods or procedures. The receipt of remuneration from patents or copyrights tempts the owners thereof to retard or inhibit research or to restrict the benefits derivable therefrom to patients, the public or the medical profession. The acceptance of rebates on prescriptions or appliances, or of commissions from attendants who aid in the care of patients is unethical. An ethical physician does not engage in barter or trade in the appliances, devices or remedies prescribed for patients, but limits the sources of his professional income to professional services rendered the patient. He should receive his remuneration for professional services rendered only in the amount of his fee specifically announced to his patient at the time the service is rendered or in the form of a subsequent statement, and he should not accept additional compensation secretly or openly, directly or indirectly, from any other source.

The prescription or dispensing by a physician of secret medicines or other secret remedial agents, of which he does not know the composition, or the manufacture or promotion of their use is unethical.

EVASION OF LEGAL RESTRICTIONS

SEC. 7.—An ethical physician will observe the laws regulating the practice of medicine and will not assist others to evade such laws.

Chapter II. Duties of Physicians to Their Patients

STANDARDS, USEFULNESS, NONSECTARIANISM

SECTION 1.—In order that a physician may best serve his patients, he is expected to exalt the standards of his profession and to extend its sphere of usefulness. To the same end, he should not base his practice on an exclusive dogma or a sectarian system, for "sects are implacable despots; to accept their thralldom is to take away all liberty from one's action and thought."* A sectarian or cultist as applied to medicine is one who alleges to follow or in his practice follows a dogma, tenet or principle based on the authority of its promulgator to the exclusion of demonstration and scientific experience. All voluntarily associated activities with cultists are unethical. A consultation with a cultist is a futile gesture if the cultist is assumed to have the same high grade of knowledge, training and experience as is possessed by the doctor of medicine. Such consultation lowers the honor and dignity of the profession in the same degree in which it elevates the honor and dignity of those who are irregular in training and practice.

PATIENCE, DELICACY AND SECRECY

SEC. 2.—Patience and delicacy should characterize the physician. Confidences concerning individual or domestic life entrusted by patients to a physician and defects in the disposition or character of patients observed during medical attendance should never be revealed unless their revelation is required by the laws of the state. Sometimes, however, a physician must determine whether his duty to society requires him to employ knowledge, obtained through confidences entrusted to him as a physician, to protect a healthy person against a communicable disease to which he is about to be exposed. In such instance, the physician should act as he would desire another to act toward one of his own family in like circumstances. Before he determines his course, the physician should know the civil law of his commonwealth concerning privileged communications.

*Nicon, father of Galen.

PROGNOSIS

SEC. 3.—The physician should neither exaggerate nor minimize the gravity of a patient's condition. He should assure himself that the patient, his relatives or his responsible friends have such knowledge of the patient's condition as will serve the best interests of the patient and the family.

THE PATIENT MUST NOT BE NEGLECTED

SEC. 4.—A physician is free to choose whom he will serve. He should, however, respond to any request for his assistance in an emergency or whenever temperate public opinion expects the service. Once having undertaken a case, the physician should not neglect the patient, nor should he withdraw from the case without giving notice to the patient, his relatives or his responsible friends sufficiently long in advance of his withdrawal to allow them to secure another medical attendant.

Chapter III. Duties of Physicians to Each Other and to the Profession at Large

Article I.—Duties to the Profession

UPHOLDING THE HONOR OF THE PROFESSION

SECTION 1.—A physician is expected to uphold the dignity and honor of his vocation.

MEMBERSHIP IN MEDICAL SOCIETIES

SEC. 2.—For the advancement of his profession, a physician should affiliate with medical societies and contribute of his time, energy and means so that these societies may represent the ideals of the profession.

SAFEGUARDING THE PROFESSION

SEC. 3.—Every physician should aid in safeguarding the profession against admission to it of those who are deficient in moral character or education.

SEC. 4.—A physician should expose, without fear or favor, incompetent or corrupt, dishonest or unethical conduct on the part of members of the profession. Questions of such conduct should be considered, first, before proper medical tribunals in executive sessions or by special or duly appointed committees on ethical relations, provided such a course is possible and provided, also, that the law is not hampered thereby. If doubt should arise as to the legality of the physician's conduct, the situation under investigation may be placed before officers of the law, and the physician-investigators may take the necessary steps to enlist the interest of the proper authority.

Article II.—Professional Services of Physicians to Each Other

DEPENDENCE OF PHYSICIANS ON EACH OTHER

SECTION 1.—As a general rule, a physician should not attempt to treat members of his family or himself. Consequently, a physician should cheerfully and without recompense give his professional services to physicians or their dependents if they are in his vicinity.

COMPENSATION FOR EXPENSES

SEC. 2.—When a physician from a distance is called to advise another physician about his own illness or about that of one of his family dependents, and the physician to whom the service is rendered is in easy financial circumstances, a compensation that will at least meet the traveling expenses of the visiting physician should be proffered him. When such a service requires an absence from the accustomed field of professional work of the visitor that might reasonably be expected to entail a pecuniary loss, such loss may, in part at least, be provided for in the compensation offered.

ONE PHYSICIAN IN CHARGE

SEC. 3.—When a physician or a member of his dependent family is seriously ill, he or his family should select

one physician to take charge of the case. The family may ask the physician in charge to call in other physicians to act as consultants.

Article III.—Duties of Physicians in Consultation

CONSULTATIONS SHOULD BE ENCOURAGED

SECTION 1.—In case of serious illness, especially in doubtful or difficult conditions, the physician should request consultations.

CONSULTATION FOR PATIENT'S BENEFIT

SEC. 2.—In every consultation, the benefit to the patient is of first importance. All physicians interested in the case should be candid with the patient, a member of his family or a responsible friend.

PUNCTUALITY

SEC. 3.—All physicians concerned in consultations should be punctual. When, however, one or more of the consultants are unavoidably delayed, the one who arrives first should wait for the others for a reasonable time, after which the consultation should be considered postponed. When the consultant has come from a distance, or when for any other reason it will be difficult to meet the physician in charge at another time, or if the case is urgent, or it be the desire of the patient, his family or his responsible friends, the consultant may examine the patient and mail his written opinion, or see that it is delivered under seal to the physician in charge. Under these conditions, the consultant's conduct must be especially tactful; he must remember that he is framing an opinion without the aid of the physician who has observed the course of the disease.

PATIENT REFERRED TO CONSULTANT

SEC. 4.—When a patient is sent to a consultant and the physician in charge of the case cannot accompany the patient, the physician in charge should provide the consultant with a history of the case, together with the physician's opinion and outline of the treatment, or so much of this as may be of service to the consultant. As soon as possible after the consultant has seen the patient he should address the physician in charge and advise him of the results of the consultant's investigation. The opinions of both the physician in charge and the consultant are confidential and must be so regarded by each.

DISCUSSIONS IN CONSULTATION

SEC. 5.—After the physicians called in consultation have completed their investigations, they and the physician in charge should meet by themselves to discuss the course to be followed. Statements should not be made nor should discussion take place in the presence of the patient, his family or his friends, unless all physicians concerned are present or unless all of them have consented to another arrangement.

RESPONSIBILITY OF ATTENDING PHYSICIAN

SEC. 6.—The physician in charge of the case is responsible for treatment of the patient. Consequently, he may prescribe for the patient at any time and is privileged to vary the treatment outlined and agreed on at a consultation whenever, in his opinion, such a change is warranted. However, after such a change, it is best to call another consultation; then the physician in charge should state his reasons for departing from the course decided at the previous conference. When an emergency occurs during the absence of the physician in charge, a consultant may assume authority until the arrival of the physician in charge, but his authority should not extend further without the consent of the physician in charge.

CONFLICT OF OPINION

SEC. 7.—Should the physician in charge and a consultant be unable to agree in their view of a case, another consultant should be called or the differing consultant should withdraw However, since the patient employed the consultant to obtain his opinion, he should be permitted to state it to the patient, his relative or his responsible friend, in the presence of the physician in charge.

CONSULTANT AND ATTENDANT

SEC. 8.—When a physician has acted as consultant in an illness, he should not become the physician in charge in the course of that illness, except with the consent of the physician who was in charge at the time of the consultation.

Article IV.—Duties of Physicians in Cases of Interference

MISUNDERSTANDINGS TO BE AVOIDED

SECTION 1.—A physician, in his relationship with a patient who is under the care of another physician, should not give hints relative to the nature and treatment of the patient's disorder; nor should a physician do anything to diminish the trust reposed by the patient in his own physician. In embarrassing situations, or whenever there seems to be a possibility of misunderstanding with a colleague, a physician should seek a personal interview with his fellow.

SOCIAL CALLS ON PATIENT OF ANOTHER PHYSICIAN

SEC. 2.—When a physician makes social calls on another physician's patient he should avoid conversation about the patient's illness.

SERVICES TO PATIENT OF ANOTHER PHYSICIAN

SEC. 3.—A physician should not take charge of, or prescribe for another physician's patient during any given illness (except in an emergency) until the other physician has relinquished the case or has been formally dismissed.

CRITICISM TO BE AVOIDED

SEC. 4.—When a physician does succeed another physician in charge of a case, he should not disparage, by comment or insinuation, the one who preceded him. Such comment or insinuation tends to lower the confidence of the patient in the medical profession and so reacts against the patient, the profession and the critic.

EMERGENCY CASES

SEC. 5.—When a physician is called in an emergency because the personal or family physician is not at hand, he should provide only for the patient's immediate need and should withdraw from the case on the arrival of the personal or family physician. However, he should first report to the personal or family physician the condition found and the treatment administered.

PRECEDENCE WHEN SEVERAL PHYSICIANS ARE SUMMONED

SEC. 6.—When several physicians have been summoned in a case of sudden illness or of accident, the first to arrive should be considered the physician in charge. However, as soon as is practicable, or on the arrival of the acknowledged personal or family physician, the first physician should withdraw. Should the patient, his family or his responsible friend wish some one other than he who has been in charge of the case, the patient or his representative should advise the personal or family physician of his desire. When, because of sudden illness or accident, a patient is taken to a hospital without the knowledge of the physician who is known to be the personal or family physician, the patient should be returned to the care of the personal or family physician as soon as is feasible.

A COLLEAGUE'S PATIENT

SEC. 7.—When a physician is requested by a colleague to care for a patient during the colleague's temporary absence, or when, because of an emergency, a physician is asked to see a patient of a colleague, the physician should treat the patient in the same manner and with the same delicacy that he would wish used in similar circumstances if the patient were his responsibility. The patient should be returned to the care of the attending physician as soon as possible.

SUBSTITUTION IN OBSTETRIC WORK

SEC. 8.—When a physician attends a woman who is in labor because the one who was engaged to attend her is absent, the physician summoned in the emergency should relinquish the patient to the first engaged, on his arrival. The one in attendance is entitled to compensation for the professional services he may have rendered.

Article V.—Disputes Between Physicians

ARBITRATION

SECTION 1.—Whenever there arises between physicians a grave difference of opinion, or of interest, which cannot be promptly adjusted, the dispute should be referred for arbitration, preferably to an official body of a component society.

Article VI.—Compensation

LIMITS OF GRATUITOUS SERVICE

SECTION 1.—Poverty of a patient, and the obligation of physicians to attend one another and the dependent members of the families of one another, should command the gratuitous services of a physician. Institutions and organizations for mutual benefit, or for accident, sickness and life insurance, or for analogous purposes, should meet such costs as are covered by the contract under which the service is rendered.

CONDITIONS OF MEDICAL PRACTICE

SEC. 2.—A physician should not dispose of his services under conditions that make it impossible to render adequate service to his patients, except under circumstances in which the patients concerned might be deprived of immediately necessary care.

CONTRACT PRACTICE

SEC. 3.—Contract practice as applied to medicine means the practice of medicine under an agreement between a physician or a group of physicians, as principals or agents, and a corporation, organization, political subdivision or individual, whereby partial or full medical services are provided for a group or class of individuals on the basis of a fee schedule, or for a salary or for a fixed rate per capita.

Contract practice *per se* is not unethical. Contract practice is unethical if it permits of features or conditions that are declared unethical in these Principles of Medical Ethics or if the contract or any of its provisions causes deterioration of the quality of the medical services rendered.

FREE CHOICE OF PHYSICIAN

SEC. 4.—Free choice of physician is defined as that degree of freedom in choosing a physician which can be exercised under usual conditions of employment between patients and physicians. The interjection of a third party who has a valid interest, or who intervenes between the physician and the patient does not *per se* cause a contract to be unethical. A third party has a valid interest when, by law or volition, the third party assumes legal responsibility and provides for the cost of medical care and indemnity for occupational disability.

COMMISSIONS

SEC. 5.—When a patient is referred by one physician to another for consultation or for treatment, whether the physician in charge accompanies the patient or not, the giving or receiving of a commission by whatever term it may be called or under any guise or pretext whatsoever is unethical.

PURVEYAL OF MEDICAL SERVICE

SEC. 6.—A physician should not dispose of his professional attainments or services to any hospital, lay body, organization, group or individual, by whatever name called, or however organized, under terms or conditions which permit exploitation of the services of the physician for the financial profit of the agency concerned. Such a procedure is beneath the dignity of professional practice and is harmful alike to the profession of medicine and the welfare of the people.

Chapter IV. The Duties of Physicians to the Public

PHYSICIANS AS CITIZENS

SECTION 1.—Physicians, as good citizens, possessed of special training, should advise concerning the health of the community wherein they dwell. They should bear their part in enforcing the laws of the community and in sustaining the institutions that advance the interests of humanity. They should cooperate especially with the proper authorities in the administration of sanitary laws and regulations.

PUBLIC HEALTH

SEC. 2.—Physicians, especially those engaged in public health work, should enlighten the public concerning quarantine regulations and measures for the prevention of epidemic and communicable diseases. At all times the physician should notify the constituted public health authorities of every case of communicable disease under his care, in accordance with the laws, rules and regulations of the health authorities. When an epidemic prevails, a physician must continue his labors without regard to the risk to his own health.

PHARMACISTS

SEC. 3.—Physicians should recognize and promote the practice of pharmacy as a profession and should recognize the cooperation of the pharmacist in education of the public concerning the practice of ethical and scientific medicine.

AMERICAN MEDICAL ASSOCIATION

AMA Mental Health Program

The American Medical Association's Committee on Mental Health, the newest scientific group of the AMA, has outlined a thirteen-point program on which individual state and county medical societies may base their mental health activities to aid in improving mental hygiene. The points as listed by Dr. Richard J. Plunkett, Chicago, secretary of the committee, follow:

1. Study medical service plans to determine ways by which they may more effectively provide for treatment of mental and emotional illness.

2. Establish citizen groups for promotion of mental health education services.

3. Recommend to legislatures increased budgets for mental institutions.

4. Serve as a central office for dissemination of information on programs for mental health to the medical profession and public.

5. Expand efforts toward establishing psychiatric depart-

ments or services in general hospitals and increasing other psychiatric services in the community.

6. Recommend to legislative committees of state medical associations that changes or amendments be made in medical practice acts to include "mental illness" and "psychotherapy" as being within the legal definition and responsibility of the practice of medicine.

7. Improve facilities and level of professional care in state institutions for care of the mentally ill.

8. Establish facilities for psychiatric consultation to courts and penal institutions.

9. Review and sponsor legislation for state mental health activities relating to problems of alcoholism and drug addiction.

10. Sponsor improved state laws for commitment to mental institutions, and provide for and encourage voluntary admissions to mental institutions.

11. Sponsor programs for postgraduate education of physicians in the utilization of psychiatric principles in the practice of medicine.

12. Sponsor programs in psychiatry and mental health in county society meetings during Mental Health Week in May of each year.

13. Encourage the setting up of collaborating committees on mental health in all county societies.

TEXAS MEDICAL ASSOCIATION

"Complete the Campaign for '53"

With reports on donations to the American Medical Education Fund incomplete, Dr. S. W. Thorn of Houston, statewide chairman for the 1953 drive for contributions, has urged that county chairmen get in touch with their medical colleagues early in December and make a final report on all gifts to him by January 1. Physicians who wish to make contributions need not wait to hear from their local AMEF representatives; they may take the initiative, calling or sending a check to the persons listed herewith, the AMEF chairmen in other societies, or Dr. Thorn.

One of the most successful local campaigns thus far reported in Texas was carried out in Harris County, donations from which total $3,330.50 this year. Part of this total was a $1,000 contribution from Dr. Stuart A. Wallace, which entitles him to a special award of merit from the national foundation. Other $1,000 contributors will be eligible for this special recognition.

Balanced against the donations made by Texas physicians to the cause of better medical education is the series of grants made to Texas colleges in July. The University of Texas Medical Branch received $27,455, Southwestern Medical School of the University of Texas $23,595, and Baylor University College of Medicine $22,490. Dr. Thorn points out that these grants far exceed the amount raised in the state.

AMEF chairmen in the larger county medical societies, who have pledge cards and can give further details about the need for funds, are as follows:

Bell: Dr. R. R. Curtis, Temple.
Bexar: Dr. George D. Reeves, San Antonio.
Dallas: Dr. Edward White, Dallas.
El Paso: Dr. R. F. Thompson, El Paso.
Galveston: Dr. E. S. McLarty, Galveston.
Harris: Dr. C. F. Jorns, Houston.
Jefferson: Dr. L. C. Powell, Beaumont.
Lubbock-Crosby: Dr. A. G. Barsh, Lubbock.
McLennan: Dr. H. R. Dudgeon, Jr., Waco.
Nueces: Dr. Y. C. Smith, Corpus Christi.
Potter: Dr. J. R. Lemmon, Amarillo.
Tarrant: Dr. J. D. Murphy, Fort Worth.
Travis: Dr. M. Allen Forbes, Austin.
Wichita: Dr. William P. Lowry, Wichita Falls.

HOTEL RESERVATION PROCEDURE

Physicians and others planning to attend the Texas Medical Association annual session in San Antonio from May 3 to 5 may write directly to the hotel of their choice after January 1 for room reservations. Reservations will be made on a first-come, first-served basis. No deposit on rooms will be required; however, cancellations should be made promptly in the event that an applicant finds he cannot attend the meeting.

In writing, the following information should be included: the type and number of accommodations desired, relationship of applicant to the Association or Auxiliary (such as committee chairman, technical exhibitor, or member of the House of Delegates), date and hour of arrival and departure, names and addresses of all persons for whom reservations are being requested, and the applicant's own name and mailing address. Each applicant should request a written confirmation from the hotel and take this confirmation to San Antonio when he registers.

The House of Delegates will meet May 2, related specialty organizations May 2 and 3, and the Association proper May 3-5. Activities of the House of Delegates are scheduled for the Gunter Hotel, and the Woman's Auxiliary will have its headquarters at the Menger Hotel. Scientific activities will be at the Municipal Auditorium, Gunter Hotel, and Plaza Hotel. Personnel in charge of technical exhibits will be housed at the Plaza Hotel.

A list of hotels and motels, their addresses, total rooms, and minimum charges follows:

HOTELS

Bluebonnet, 426 N. St. Mary's, 250 rooms, $3.
Crockett, 301 E. Crockett, 150 rooms, $3.
Gunter, 205 E. Houston, 550 rooms, $5 (Association officials).
Menger, 204 Alamo Plaza, 130 rooms, $5.50 (Auxiliary officials).
Plaza, 309 S. St. Mary's, 500 rooms, $5 (technical exhibitors).
Robert E. Lee, 311 W. Travis, 250 rooms, $3.50.
St. Anthony, 300 E. Travis, 250 rooms, $5.
White Plaza, 203 E. Travis, 204 rooms, $3.25 (Association officials).

MOTELS

Aero Motel, 3535 Broadway, 24 units, $5.
El Montan Motor Courts, 6806 San Pedro, 23 units, $6.
Park Mo-Tel, Inc., 3617 Broadway, 52 units, $4.
Ranch Motel, 3101 Broadway, 27 units, $5.
Rio Lado Drive In Hotel, 1100 N. St. Mary's, 30 rooms, $6.
Siesta Motor Courts, 4341 Fredericksburg Road, 36 units, $4.50.
The Westerner, 1116 Austin Highway, 85 rooms, $5.

CHANGES IN ASSOCIATION COMMITTEES

A number of changes in committees of the Texas Medical Association have taken place since publication of the official list in the June JOURNAL.

A newly appointed Committee for Coordination of Insurance Claims is composed of Dr. M. C. Overton, Jr., Pampa, chairman, and Drs. Tom B. Bond, Fort Worth; John H. Wootters, Houston; James W. Rainer, Odessa; and J. B. Copeland, San Antonio.

A second committee to work in the area of insurance is the Committee for Liaison with Workmen's Compensation Insurance Companies with the following membership: Dr. Sam N. Key, Jr., Austin, chairman, and Drs. Frederick C. Rehfeldt, Fort Worth; R. G. Carpenter, Dallas; M. H. Morris, San Antonio; and Wendell H. Hamrick, Houston.

Drs. A. C. Scott, Jr., Temple, and C. Forrest Jorns, Houston, have been named to serve on a joint committee to cooperate with the Board of Nurse Examiners in supporting proposed changes in the curriculum in public health and

psychiatric nursing now recommended for the accreditation of hospital schools of nursing and in designing a recruitment program for the increase of hospital personnel. Other members of the committee will represent hospital administrators, the Texas Graduate Nurses Association, and the Board of Nurse Examiners.

At the request of Dr. John F. Pilcher, Corpus Christi, he was replaced as chairman of the Committee on Public Health by Dr. Hugh C. Welsh, Houston, already a member of the committee. Dr. Pilcher continues to serve on the committee.

Dr. A. B. Cooper, El Paso, has replaced Dr. Don P. Morris, Dallas, on the Committee on Mental Health, and Dr. George D. Bruce, Baytown, has been added to the Committee on Rural Health.

Dr. Howard O. Smith, Marlin, has succeeded Dr. Clifford G. Swift, Cameron, as vice-councilor of District 12, and Dr. L. J. Webster, Abilene, has succeeded Dr. H. H. Cartwright, Breckenridge, as vice-councilor of District 13.

Workmen's Compensation Studied

Announcement is made elsewhere in this section of the appointment of a Texas Medical Association Committee on Liaison with Workmen's Compensation Insurance Companies. This group with representatives from insurance companies met October 23 to formulate objectives for the joint Physicians and Carriers Workmen's Compensation Committee; the joint committee plans to meet again January 15.

The objectives agreed upon follow:

1. To encourage the use of the grievance committee or a special insurance committee for the settlement of any complaint arising between physicians and insurance carriers in the care of workmen's compensation cases. The Physicians and Carriers Workmen's Compensation Committee will act in an advisory capacity to the various local committees.

2. To act as a grievance body to review any complaint or decision when requested by a physician or insurance carrier.

3. To discuss at the joint committee meetings the problems which beset each group. The group proposes to see that the provisions of the Workmen's Compensation Insurance Law are properly carried out by the interested parties.

4. To act as an educational body for the dissemination of information of interest to both parties.

5. To play an important function in the clarification of the many problems that arise with the care of injured workmen, in solving any dispute between each group, and in serving as a public relations committee to the general public.

Dr. Sam N. Key, Jr., Austin, chairman of the physicians' group; Dr. Frederick C. Rehfeldt, Fort Worth; and Dr. M. H. Morris, San Antonio, were present for the initial meeting of the joint committee as were T. W. Wassell, Dallas (Texas Employers' Insurance Association), chairman of the insurance group; George K. Hempel, Dallas (Travelers Insurance Company), vice-chairman; John W. DuLaney, Dallas (Liberty Mutual Insurance Company), secretary; Charles E. Granger, Dallas (American Surety Company); L. R. Monday, Houston (American General Insurance Company); G. H. Sawyer, Dallas (Continental Casualty Company); and Roy Leach, Longview, president of the Independent Adjusters Association of Texas. The insurance men present represented the Association of Casualty and Surety Companies, National Association of Mutual Casualty Companies, Association of Texas Fire and Casualty Executives, independent companies, and the Independent Adjusters Association of Texas. Together, the companies represented account for substantially all of the workmen's casualty insurance written in Texas, and only this type of insurance and the problems of medical care which go with it will be considered by the committee.

EXECUTIVE COUNCIL MEETS JANUARY 22-24

A highlight of the week-end of the Executive Council meeting, January 22-24 in Austin at Texas Medical Association headquarters, will be the Presidents and Secretaries Conference January 23. Dr. Frank E. Wilson, director of the American Medical Association's Washington office, will be a featured speaker.

The first session of the Presidents and Secretaries Conference, scheduled for 10 a. m., will be devoted to reports of chairmen of the key boards, councils, and committees of the Association. The second session, beginning at 2 p. m., will include the address "Who and What We Are Discussing" by Dr. Wilson, also "Medicine's Policy and What It Means" by Dr. Everett C. Fox, Dallas, and "Implementing the Policy" by Dr. F. J. L. Blasingame, Wharton. Both sessions will end with question and answer periods.

Many of the councils and committees of the Association will meet either January 22 or 23. The Executive Council will convene at 8:30 a. m. January 24.

COUNTY SOCIETIES

Angelina County Society

October 13, 1953

(Reported by Byford H. Denman, President)

The Angelina County Medical Society met October 13 at radio station KTRE in Lufkin to hear the telephone postgraduate broadcast on "Obstetric Emergencies" presented by the Texas Medical Association.

November 10, 1953

(Reported by Byford H. Denman, President)

Preceding the telephone broadcast on antibiotics to which the Angelina County Medical Society listened for its program November 10, members of the society, meeting in Lufkin, elected officers for the year 1954. Named to take office January 1 are three Lufkinites: Gail Medford, president; C. W. Evans, vice-president; and F. C. Orman, secretary-treasurer. A committee was appointed to plan a dinner in January to honor these officers and those who have been serving this year: Byford H. Denman, president; Dr. Medford, vice-president; and Dan Spivey, secretary-treasurer.

The society also voted to subscribe to *Today's Health* for the local high schools and library.

Brazoria County Society

September 24, 1953

(Reported by Carlos E. Fuste, Jr., Secretary)

The Brazoria County Medical Society heard Stanley Olsen, dean of Baylor University College of Medicine, Houston, discuss administrative problems encountered in medical education at its monthly meeting September 24 in Freeport. Twenty-two members attended.

It was announced that American Medical Association exhibits, sponsored by the Brazoria County Medical Society, were to be on display at the Brazoria County Fair October 6-10.

Oscar L. Johnson and Wayne K. Steele were unanimously accepted for membership.

October 29, 1953

(Reported by Carlos E. Fuste, Jr., Secretary)

Surgical Management of Carcinoma of Head and Neck—James Allen Chamberlin, Houston.

A Houston guest presented the scientific program for the dinner meeting of Brazoria County Medical Society at Free-

port, October 29. The society considered the possibility of placing with one or more television stations of the area the television films released by the American Medical Association.

Cameron-Willacy Counties Society

October 19, 1953

(Reported by David F. Nickell, Secretary)

Some Interesting Facts on Tuberculosis—R. C. Bernard, Harlingen.

Members of the Cameron-Willacy Counties Medical Society and Auxiliary met together October 19 in San Benito for dinner and a paper on tuberculosis. The society meeting independently then held a business session, during which permission was granted for a tuberculosis survey in Cameron County, approval was given of plans for special newspaper sections commemorating the hundredth anniversary of organized medicine in Texas, and announcement was made of the desirability of making contributions to medical schools through the American Medical Education Foundation.

Cass-Marion Counties Society

September 9, 1953

Use and Abuse of Antibiotics—Clarence H. Webb, Shreveport.

Nineteen physicians and their wives met for dinner in Linden September 9, following which the Cass-Marion Counties Medical Society heard an address by a Shreveport physician. The Auxiliary met separately for its own program.

Dallas County Society

October 13, 1953

(Reported by W. W. Fowler, Secretary)

ACTH, Cortisone, and Concept of Potassium Deficiency—Donald W. Seldin, Dallas.

The above discussion was presented by Dr. Seldin at a joint meeting of the Dallas County Medical Society and the St. Paul Hospital medical staff October 13 at St. Paul Hospital in Dallas.

After entertainment by the hospital's Nurses Glee Club; a discussion of medical economic news by Mr. Millard Heath, executive secretary of the medical society; and a brief business meeting of the St. Paul staff, Jack G. Kerr, president of the society, conducted a business session. The following doctors were elected to membership: John Arthur Stuart, Jr., John Alfred Emmert, Marcus B. Dalkowitz, Glenn R. Cherry, Mary Sabo Hoffman, Ellen Loeb, and Robert Edward Speegle. Also, the following members were elected to military membership: Bob Smith, George Thomas Shires, Ben K. Howard, Gerald G. Mullikin, Charles Harry Lodowski, M. C. Jackson, and John L. Baskin.

Hardeman-Cottle-Foard-Motley Counties Society

September 24, 1953

(Reported by J. M. Taylor, Secretary)

Common Urologic Conditions Confronted in General Practice—J. R. Reagan, Wichita Falls.

Early Diagnosis of Carcinoma of Stomach—O. H. Trimble, Wichita Falls.

Two Wichita Falls physicians presented papers at the September 24 meeting of Hardeman-Cottle-Foard-Motley Counties Medical Society in Paducah. Nine members heard the discussions and enjoyed a steak dinner.

Harrison County Society

September 11, October 9, November 12, December 11, 1953

Diagnosis and Cure of Pheochromocytoma—Nicholas C. Hightower, Temple.

Medical and Surgical Treatment of Burns—Willard C. Sellman, Dallas.

What's Cooking in Medicine?—R. B. Robins, Camden, Ark.

Diagnosis and Treatment of Thyroid Diseases—C. F. Hamilton, Dallas.

A series of four monthly addresses, the topics and speakers for which are listed above, have been presented by the Harrison County Medical Society for physicians of East Texas and West Louisiana in an effort to bring to them firsthand information of new medical techniques developed by distinguished specialists. The first two and last of the lectures were presented at luncheon meetings in Marshall; the third at an evening meeting open to wives of the doctors.

Arrangements for the series were made by Roger Harmon, Maurice Murphy, and Samuel Tenney.

Lubbock-Crosby Counties Society

October 1, 1953

(Reported by Frank C. Goodwin, Secretary)

Problem of Cancer Control—Mr. J. Louis Neff, Austin.

Mr. Neff, executive secretary of the Texas Division of the American Cancer Society, discussed the above subject at the October 1 meeting of the Lubbock-Crosby Counties Medical Society in Lubbock. The society voted to support the program outlined by Mr. Neff for the establishment of a local cancer society chapter. The motion was made by James G. Morris and seconded by Charles H. Nash, Jr.

Miss Katherine Daniel, field director of the Texas Cancer Society, was present at the meeting and was introduced by A. G. Barsh.

Wallace I. Hess, president, presided over the meeting.

October 6, 1953

(Reported by Frank C. Goodwin, Secretary)

Current Concepts in Treatment of Hypertension—Arthur Grollman, Dallas.

Discussion—Roy Sheffield, John Selby, O. Brandon Hull, Lubbock.

In addition to a lively discussion of hypertension after a paper on the subject by Arthur Grollman, professor of experimental medicine at Southwestern Medical School, Dallas, members of the Lubbock-Crosby Counties Medical Society at their meeting October 6 in Lubbock considered a variety of business.

Favorable reaction was evident to the proposal that the society cooperate in establishing a health exhibit at the museum at Texas Technological College. Dr. Bruno Gebhard, director of the Cleveland Health Museum, was to be in Lubbock later in the month to discuss the possibility.

Allen T. Stewart reminded the society of its agreement to spend $500 for furnishings and decorations and $100 annually for periodicals in return for reading space in the new library building. He also reported that some insurance claim adjusters had raised some problems about hospital and physician charges, and he, Frank B. Malone, and John Selby were appointed by the president to discuss these problems with the adjusters.

Potter County Society

October 12, 1953

(Reported by Mrs. Cassie Atherton, Executive Secretary)

Management of Cutaneous Infections—Clarence S. Livingood, Galveston.

A brief business session of the Potter County Medical Society followed a dinner and the above discussion by Dr. Livingood October 12 at Amarillo. The society went on record as approving the essay contest sponsored by the Association of American Physicians and Surgeons; the amount to be given for local prizes is to be determined later.

Woolworth Russell announced that a seminar sponsored by the Academy of General Practice would be held October 21 in Amarillo.

Dr. Livingood, who was introduced by William E. Laur, illustrated his lecture with colored slides.

November 9, 1953

(Reported by Mrs. Cassie Atherton, Executive Secretary)

Legal Aspects of Physician's Practice in Relation with Patients—Mr. Lewis F. Russell, Dallas.

A Dallas attorney was speaker at a dinner meeting of Potter County Medical Society, November 9 in Amarillo, at which forty-seven members were present. The society voted to allocate $200 for prizes for use by the auxiliary in the essay contest sponsored in the local high schools as a part of the national contest of the Association of American Physicians and Surgeons. It also voted to amend the constitution and by-laws to provide for the election of a president-elect and agreed to install newly elected officers at the January meeting each year. Plans for the Third District Medical Society meeting April 13 were discussed.

Tarrant County Society

September 23, 1953

The Tarrant County Medical Society met September 23 in Fort Worth in conjunction with the tumor clinic of the Southwest Regional Cancer Conference. One hundred members of the society attended.

September 29, 1953

(Reported by S. W. Wilson, Secretary)

A special meeting of the Tarrant County Medical Society was called September 29 in Fort Worth to consider the offer of the Fort Worth Academy of Medicine to establish the office and meeting place for the society in the academy's new building. Porter Brown, a member of the board of directors of the academy, explained the history, progress, aims, and other points of interest about the academy. May Owen, secretary-treasurer of the academy, then presented a resolution calling for the transfer of the office, library, and properties of the Tarrant County Medical Society from the Medical Arts Building to the academy building at 3855 Tulsa Way and expressing appreciation to the Jesse Jones Interests for the use of space so generously provided by them for many years. The motion was unanimously adopted by the 116 members present.

October 20, 1953

(Reported by S. W. Wilson, Secretary)

Extensive amendments to the constitution and by-laws were presented by Gatlin Mitchell, chairman of the constitution and by-laws committee, when forty-three members of Tarrant County Medical Society met October 20 in Fort Worth. It was announced that these amendments would be voted on at the next meeting. The society adopted a resolution of thanks to Mr. Amon G. Carter and the Amon G. Carter Foundation for their generosity in erecting and furnishing a library, museum, and meeting hall for the physicians of Fort Worth and paid tribute to the late Dr. J. Hyal Brown.

November 3, 1953

(Reported by S. W. Wilson, Secretary)

A resolution urging Congress to amend the Veterans Administration Act to provide medical and hospital care only for service connected disabilities was adopted November 3 by the Tarrant County Medical Society meeting in Fort Worth. The society adopted a series of amendments to its constitution and by-laws and heard several additional proposed amendments. Approval was given to a survey by the Fort Worth Quartermaster Depot of its civilian employees in an effort to determine the extent of diseases of the lungs.

Tom Green-Eight County Society

September 8, 1953

(Reported by T. G. Coleman, Secretary)

Gordon Madding, president of the Tom Green-Eight County Medical Society, presided over the society's regular monthly meeting held September 8 in San Angelo. Thirty-nine members attended.

After Mr. John W. Graham explained the operation of the San Angelo Medical and Dental Service Bureau, which includes a telephone answering service and collection and credit information service, the society voted to go on record as approving this bureau.

A motion that physicians make minimum charges for injuries received in high school athletics and also give a 25 per cent discount was passed, and the following committee was appointed to see that a doctor is on the bench at all games: H. M. Anderson, Cecil M. French, and Gordon A. Pilmer.

The society agreed to underwrite the national essay contest sponsored by the Association of American Physicians and Surgeons and to offer prizes of $100, $50, and $25 for local winners.

Francis Spencer, Royce W. Pruet, and J. W. Singleton were elected to military membership.

October 13, 1953

(Reported by T. G. Coleman, Secretary)

A brief business session of the Tom Green-Eight County Medical Society was presided over by the vice-president, Joe L. Cornelison, October 13 in San Angelo. The nineteen members present then listened to the telephone broadcast on obstetric emergencies presented by the Texas Medical Association.

Travis County Society

September 15, 1953

(Reported by C. H. McCuistion, Secretary)

The first fall meeting of the Travis County Medical Society was held September 15 at the Texas Medical Association Library and Headquarters Building in Austin. The meeting was devoted entirely to business matters, and such things were discussed as listing of physicians in the yellow pages of the telephone directory, membership in the Association of American Physicians and Surgeons, the American Medical Education Foundation, the society's participation in the observance of the Texas Medical Association's centennial anniversary, and contributions of local physicians toward construction at a local hospital. Committee reports were made.

It was announced that Herbert L. Shepard and Matthew F. Kreisle, Jr. were elected as new society members. Also, Robert Franken transferred his membership from the Bexar County Medical Society to Travis County.

Announcement also was made of the change in meeting dates for October and November to take advantage of the postgraduate telephone broadcasts sponsored by the Texas Medical Association. The subject for October 13 was "Obstetric Emergencies" with William F. Mengert, Dallas, as moderator. James E. Kreisle, Austin, was scheduled to moderate the November 10 meeting on "Use of Antibiotics."

Webb-Zapata-Jim Hogg Counties Society

October 6, 1953

(Reported by A. S. McGee, Secretary)

Epilepsy and Its Equivalents in General—Philip Magrish, San Antonio.

After a dinner at the Plaza Hotel in Laredo October 6, the Webb-Zapata-Jim Hogg Counties Medical Society held a business session and heard the above discussion by Dr.

Magrish, who also showed a number of electro-encephalograms.

Margaret Cigarroa announced that preparations had been made for the society to participate in the October 13 telephone postgraduate broadcast sponsored by the Texas Medical Association.

Nineteen members and four guests were present, and A. C. King, president, presided.

Wharton-Jackson-Matagorda-Fort Bend Counties Society

November 10, 1953

(Reported by Lorraine I. Stengl, Secretary)

Surgical Considerations in Treatment of Cardiac Disease—Michael E. DeBakey, Houston.

Colored slides and a three dimension film were used by Michael E. DeBakey, member of the faculty at Baylor University College of Medicine, Houston, in his presentation on cardiac disease for members of the Wharton-Jackson-Matagorda-Fort Bend Counties Medical Society and Auxiliary in Bay City on November 10. Thirty-nine were in attendance.

DISTRICT SOCIETIES

Fourth District Society

October 8, 1953

(Reported by P. M. Wheelis, Secretary)

Testing Function of Adrenal Gland—James A. Greene, Houston.
Use of Cortisone and ACTH in Treatment of Blood Dyscrasias—Robert A. Hettig, Houston.
Roentgen-Ray Diagnosis of Peptic Ulcers—Arno W. Sommer, Temple.
Mechanism of Pain in Duodenal Ulcers—N. C. Hightower, Temple.
Will Private Practice of Medicine Survive?—F. J. L. Blasingame, Wharton.
Radioisotopes in Relation to Thyroid Diseases—Herbert Allen, Houston.
Operation of Tumor Clinic in Small Community—Jarrett E. Williams, Abilene.
Roentgen-Ray Diagnosis of Lesions of Colon—Dr. Sommer.
Newer Concept of Cardiospasm—Dr. Hightower.

After the above scientific program and luncheon address by Dr. Blasingame at the annual meeting of the Fourth District Medical Society in Brownwood October 8, a business session was held, during which the following new officers were elected: Paul M. Wheelis, Brownwood, president; James P. Anderson, Brady, vice-president; and James N. White, San Angelo, secretary.

The Rev. Hubert Smith, Brownwood, delivered the invocation, and Mr. Smith Bell, mayor of Brownwood, gave an address of welcome.

Fifty-seven physicians attended.

Eighth District Society

October 8, 1953

(Reported by George E. Glover, Jr., Secretary)

Vascular Nevi—J. Fred Mullins, Galveston.
Diagnosis and Treatment of Carcinoma of Pancreas—Edward B. Rowe, Galveston.
Symposium of Lymphoma—Caroline Rowe, Edward B. Rowe, John C. Meadows, Jr., and John H. Childers, all of Galveston.

The above scientific program, arranged under the direction of the John Sealy Hospital Tumor Clinic, Galveston, was presented at the meeting of the Eighth District Medical Society, held October 8 in Wharton in conjunction with the district meeting of the American Cancer Society.

Mr. W. E. Syers, Austin, public relations counsel of the Texas Medical Association, gave an address at the dinner session.

The following officers were elected: Howard Z. Fretz, Wharton, president; George E. Glover, Jr., Victoria, secretary-treasurer; and James H. Wooten, Jr., Columbus, renominated for district councilor.

Thirty-four members of the medical society attended.

Eleventh District Society

October 29, 1953

(Reported by Marlin T. Braswell, Secretary)

Silent Urinary Tract Lesions—Hugh F. Rives, Jacksonville.
Veterans' Problems and the Doctor—George Turner, El Paso.
Recent Developments in Cardiovascular Surgery with Particular Reference to Use of Vascular Homografts—Denton A. Cooley, Houston.
General Practice in Relation to Obstetrics—C. Forrest Jorns, Houston.

Forty-eight members attended the afternoon meeting of the Eleventh District Medical Society, October 29 in Henderson, hearing the program outlined above. After the scientific session, officers were elected as follows: president, John Dean, Crockett; vice-president, T. M. Jarmon, Tyler; secretary-treasurer, Marlin T. Braswell, Henderson; directors, Loyd Deason, Henderson; Harvey Bell, Palestine; and F. E. Felder, Palestine.

Members of the society and auxiliary were entertained at dinner by a talk by Mr. Peppy Blount, Tyler, former member of the Legislature.

The next meeting of the society will be held in Crockett in the spring.

AUXILIARY SECTION

MENTAL HEALTH, A NEW PROJECT

Health education long has been one of the chief objectives of the Woman's Auxiliary. Its program calls for fact finding by members of the organization, education of the public, and action to improve existing conditions as need is determined by study. In its infancy, the Auxiliary in Texas was concerned with such topics as adequate sanitation in the handling and distribution of milk. More recently, geriatrics, civil defense relating to medical care, safety, and other current problems have come within the scope of the Auxiliary. One of the most recently added committees in this general field relates to mental health, a subject of such urgent concern in these times that the American Medical Association has established a Committee on Mental Health and encouraged its Woman's Auxiliary to do so.

Mrs. A. B. Pumphrey, Fort Worth, chairman of the Committee on Mental Health of the Woman's Auxiliary to the Texas Medical Association, points out that one of the first steps in educating members of the Auxiliary relative to mental health problems is to ascertain the facts about the mental health institutions in Texas and the conditions which lead patients to them. Personnel—training, interest, and number; physical facilities—housing, laboratories, out-patient clinics; guidance help—all these are important factors to consider. It should be remembered, too, that childhood problems contribute to mental distress which may not become evident until adulthood.

Officers of the Woman's Auxiliary to the Texas Medical Association: President, Mrs. E. W. Coyle, San Antonio; President-Elect, Mrs. Mark H. Latimer, Houston; First Vice-President (Organization), Mrs. John D. Gleckler, Denison; Second Vice-President (Physical Examinations), Mrs. Cecil O. Patterson, Dallas; Third Vice-President (Today's Health), Mrs. Ralph B. Payne, Amarillo; Fourth Vice-President (Program), Mrs. Guy Knolle, Houston; Recording Secretary, Mrs. Paul Brindley, Galveston; Corresponding Secretary, Mrs. Charles L. McGehee, San Antonio; Treasurer, Mrs. Oscar M. Marchman, Jr., Dallas; Publicity Secretary, Mrs. Allen H. Neighbors, Jr., Austin;

In the United States mental patients occupy more hospital beds than all other patients combined, Mrs. Pumphrey reiterates. It is estimated that mental illnesses and personality factors affect 6 per cent, 1 out of 16, or 9,000,000 people in this country. About 1,000,000 persons receive treatment each year in mental hospitals.

In Texas the mental hospitals all are overcrowded and understaffed as to physicians, nurses, attendants, social workers, and clinical psychologists. Legislative appropriations, Mrs. Pumphrey indicates, now amount to about $2 per day per patient whereas the national standard is $5.

"Why should we care about mental health?" Mrs. Pumphrey asks. "Because we are interested in health in general, the well-being of our fellow man, a safe and sane population in our communities and on the road, and the tremendous tax funds necessary for care of mental patients. There is a staggering social cost of crime, delinquency, suicide, alcoholism, drug addiction, divorce, and child problems. We are parents, taxpayers, and good citizens. We are concerned with the darkening threat of mental illness, and as Auxiliary members we will help to shape public opinion as well as our own to do more to overcome it. If this problem is attacked by concerted, sustained, and intelligent effort, there is no doubt much mental illness can be alleviated. We can work with doctors, mental health societies, guidance clinics, and legislators to improve mental health and the forces which would destroy it."

COUNTY AUXILIARIES

The problems of youth held the spotlight in two recent county auxiliary meetings. The *Hopkins-Franklin Counties Auxiliary,* meeting November 4 in the home of Mrs. Joseph Longino, Sulphur Springs, considered "Youth and Narcotics." Mrs. Royal Ramey pointed out that addiction to narcotic drugs is a nationwide problem. She explained that for most youthful addicts the urge to conform to group behavior is one of the main contributing factors. Mrs. Ramey reminded that one of two United States Public Health Service hospitals in which treatment of narcotic addicts is carried out is in Fort Worth.

"Juvenile Delinquency" was the subject of Hermon Pipkin, Amarillo attorney, at the October 13 meeting of *Potter County Auxiliary.* Mr. Pipkin stated that today no child commits a real crime until he is of age, but that what is now called delinquency is an outgrowth of what used to be called crime. He expressed the opinion that correct parental supervision before the child is 12 years of age and "the will to do right" hold the solution. Mrs. W. C. Dine, program chairman, introduced the speaker to about fifty persons.

Community service was the theme of several recent activities of auxiliaries. *Nacogdoches County Auxiliary* held a silver tea November 4 in the home of Mrs. G. F. Middlebrook, Nacogdoches, with proceeds to go toward the purchase of air conditioners for the City Memorial Hospital.

Efforts to relieve the nursing shortage were foremost in the thoughts of the *Dallas County Auxiliary* during September 6-13, the week designated by the mayor as Nurse Recruitment Week. Spearheaded by a committee of which Mrs. Joseph L. Knapp is chairman and Mrs. D. P. Laugenour co-chairman, the auxiliary suggested that high school students interested in the possibility of nursing as a career make certain of the subjects necessary for acceptance in a school of nursing before completing their high school courses. Student nurse uniforms were placed on display during the special week, and the auxiliary announced plans to organize Future Nurses Clubs in the vicinity.

An open meeting brought fifty representatives of leading El Paso women's organizations to a luncheon meeting of *El Paso County Auxiliary* on November 9. The visitors were invited to borrow health films through the auxiliary, and brochures on "Better Health—A Project in Living" were distributed. The program, presented under the direction of Mrs. Celso C. Stapp, public relations chairman, consisted of a motion picture on the care of the skin, "The Scrub Game," and a commentary on the film and "The Basis of Glamor" by Dr. H. D. Garrett, a dermatologist. Eighty-five members were present for the program and the luncheon, for which Mrs. Clement C. Boehler and Mrs. Gray E. Carpenter were in charge.

A year of service emphasizing nurse recruitment, civil defense, and aid to medical schools through the American Medical Education Foundation was opened by *Harris County Auxiliary* with a tea given by the executive board in the home of Mrs. C. Forrest Jorns, Houston, on September 28. With a membership of 635, the Harris organization is building its activities this year around the theme "to look up and not down, to look forward and not back, to look out and not in, and to lend a hand." Mrs. Jacob F. Schultz, president, headed the receiving line; several past presidents alternated at the tea table.

McLennan County Auxiliary featured the services of the medical profession to the community in its exhibit at the Heart o' Texas Fair in Waco the latter part of September. Care of indigents, donations of medical service to clinics, and being on call at local hospitals around the clock were mentioned in posters at the exhibit, which used as a drawing card a plastic skeleton, complete in every detail. Mrs. Eldon Fine, president, and Mrs. J. M. Garrett had charge of arranging the exhibit. Members of the auxiliary and student nurses from two local hospitals were hostesses throughout the fair. The auxiliary suspended its concentration on the exhibit to have a style show for its initial meeting of the year September 23. Mrs. Maurice C. Barnes was mistress of ceremonies for the show, for which members of the auxiliary were models.

First meetings of the year have been held by the *Cherokee County Auxiliary,* September 29 in Jacksonville, and by the *Camp-Morris-Titus Counties Auxiliary,* September 30 in Mount Pleasant. Mrs. E. W. Coyle, San Antonio, President of the Woman's Auxiliary to the Texas Medical Association, was guest speaker at the former meeting, a dinner at which Mesdames J. M. Travis, R. T. Travis, and L. L. Travis were hostesses. Mrs. D. C. Simmons, Kilgore, council woman for the Fifteenth District, and Mrs. John Hill, Marshall, member of the American Medical Education Foundation Committee of the State Auxiliary, spoke to the group in Mount Pleasant.

The *Cass-Marion Counties Auxiliary* joined members of the medical society at a banquet in Linden on September 9 and then convened in the home of Mrs. C. E. Davis for a discussion led by Mrs. Jesse DeWare and Mrs. W. S. Terry, both of Jefferson.

The members of *Tarrant County Auxiliary* are remembering with delight a Halloween costume dance and breakfast held in Fort Worth under sponsorship of the entertainment committee. Mrs. Coyle, the State President, came to Fort Worth from San Antonio to speak at the Friday 13 meeting of the auxiliary in November.

DISTRICT AUXILIARIES

Eighth District Auxiliary

Approximately fifty members of the Eighth District Auxiliary representing five of the six component county auxiliaries attended the October 23 meeting in El Campo. Mrs. E. W. Coyle, San Antonio, President of the State Auxiliary, was guest speaker at the luncheon. Past presidents and mem-

bers of the State Executive Board were recognized and plans of the various county presidents were reported. *Bulletin* subscriptions, American Medical Education Foundation contributions, essay contest participation, and yearbook production and distribution received special consideration. By-laws were adopted by the organization with Mrs. W. D. Nicholson, Freeport, president, presiding, and Mrs. J. C. Much, Richmond, acting as secretary.

Tenth District Auxiliary

The Port Arthur Chapter of Jefferson County Auxiliary entertained members of the Tenth District Auxiliary at a tea October 3 in Port Arthur at the home of Mrs. L. C. Heare. Mrs. Mark H. Latimer, Houston, President-Elect of the State Auxiliary, was an honor guest. Mrs. R. C. Bellamy, Liberty, presided over a business session. The women joined members of the district medical society for dinner.

DEATHS

W. R. HOUSTON

Dr. William R. Houston, Austin, Texas, died August 31, 1953, three days after observing his eighty-first birthday anniversary.

The son of missionary parents, Dr. Houston was born in 1872 in Hangchow, China. He returned to this country to complete his education, receiving a master of arts degree from Hampden-Sydney College in Virginia and in 1902 his doctor of medicine degree from the University of Georgia at Augusta. Later he studied in Berlin. Dr. Houston began practice in Augusta, where he subsequently became professor of medicine at his alma mater. For a time Dr. Houston taught medicine at Yale University, New Haven, and also at the Yale affiliate in Changsha, China. After four years in China, Dr. Houston with other United States nationals was requested by the United States government to leave because of civil uprisings, and he returned to the faculty of the University of Georgia. In 1935 Dr. Houston moved to Austin to open a practice from which he retired about five years later.

An honorary member of the Texas Medical Association through Travis County Medical Society, Dr. Houston was affiliated with the American Medical Association, American College of Physicians, American Clinical and Climatological Association, and Southern Interurban Clinical Club. He served the Travis County Society as president in 1939-1940 and was on the governing board of the College of Physicians. Dr. Houston was recognized as a scholar and writer whose interests extended the breadth of medical science.

Hale Houston of Austin, a brother, and his children survive Dr. Houston.

R. E. FORRESTER

Dr. Robert Early Forrester, Moran, Texas, died August 27, 1953, in that city of coronary thrombosis.

The son of the Elijah Forresters, Dr. Forrester was born August 7, 1887, in Burleson. He attended high school there and Texas Christian University in Fort Worth before receiving his medical degree from the Fort Worth School of Medicine in 1917. As a captain in the United States Army Medical Corps during World War I, he served in France and was cited for bravery beyond the call of duty. He did postgraduate study in surgery at Tulane University of Louisiana School of Medicine, New Orleans. Before moving in 1926 to Moran, where he specialized in industrial surgery until his death, Dr. Forrester practiced at Caddo, Mexia, and Corsicana.

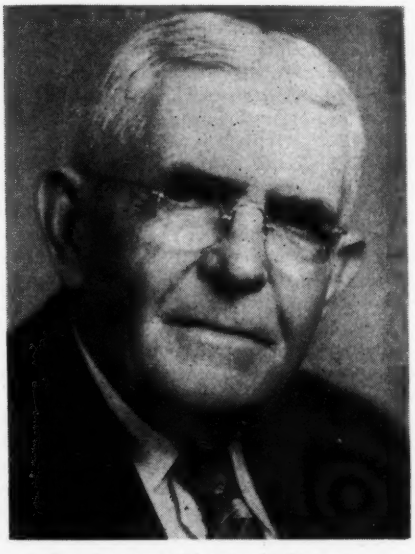
A member of the American Medical Association and the Texas Medical Association through the Stephens-Shackelford-Throckmorton Counties Medical Society, of which he was president in 1938, Dr. Forrester also was a Mason and belonged to the American Legion. He was a member of the Methodist Church.

The former Miss Jeanette Pool, whom Dr. Forrester married on December 9, 1922, in Hollywood, Calif., survives. Other survivors are a son, Robert E. Forrester, Jr., Fort Worth; a granddaughter, Aurian Forrester, Fort Worth; and two brothers, Andrew Forrester and Arthur Forrester, both of Cleburne.

A. W. DAVISSON

Dr. Alfred Williston Davisson, Corpus Christi, Texas, died in that city August 9, 1953, of cerebral hemorrhage.

Born June 1, 1867, in Peekskill, N. Y., the son of Jennie (Ennis) and Dr. Alfred Williston Davisson, Dr. Davisson attended public schools in Chicago and New York before studying for a professional career. He was a doctor of dental surgery, having received his degree from the New York College of Dentistry in 1890, and a doctor of medicine, with a degree obtained from Southern Medical College, Atlanta, Ga., in 1896. He also did postgraduate work on diseases of the eye in New York. Dr. Davisson was a dentist in Holly, N. Y., before he decided to study medicine. Then he went to Peoria, Ill., where he specialized in eye, ear, nose, and throat for seven years. In 1906 he came to Texas, settling in Corpus Christi, where he practiced medicine until a few years before his death. He served as a medical officer in the United States Army in World War I and as a medical examiner for the draft board during World War II. He also was a local physician for the Southern Pacific Railway.



DR. ALFRED WILLISTON DAVISSON

An obituary ordinarily will not be published more than four months after date of death. Cooperation in reporting deaths of physicians and in furnishing appropriate biographical material promptly is solicited.

An honorary member since 1946 of the Texas Medical Association through the Nueces County Medical Society, Dr. Davisson had belonged to the Association almost continuously since 1908. He was also a member of the American Medical Association and the Nueces County Eye, Ear, Nose, and Throat Association, which he helped to found and served twice as president. He was also president of the Nueces County Medical Society twice and secretary for six years. Dr. Davisson was a deacon in the Presbyterian Church for twenty-one years, a Rotarian for thirty-eight years. He was interested in music, and shortly after moving to Corpus Christi, he was first tenor in the Celestial City Quartet and was organizer, director, and first violinist of the orchestra which played at the opening of the Nueces Hotel. Another of Dr. Davisson's hobbies was wood working.

Dr. Davisson was the son and grandson of physicians. His grandfather studied in Philadelphia under Dr. Benjamin Rush, for whom Rush Medical College, Chicago, was named; and his father was one of the founders and first faculty members of the college.

Survivors include Dr. Davisson's wife, the former Miss Mary Irwin, whom he married September 7, 1904, in Chicago; a son, Forbes Irwin Davisson, Corpus Christi; and two grandchildren, Alfred Williston Davisson and Diana Davisson, Corpus Christi.

M. M. WALKER

Dr. Michael Muckelroy Walker, Rocksprings, Texas, died August 16, 1953, in a local hospital of pneumonia.

Born September 20, 1883, on a ranch in Lavaca County, he was the son of Sam W. and Mary (Muckelroy) Walker. He received his early schooling at Wichita Falls High School and Washington University, St. Louis, and was graduated with the degree of doctor of medicine from the St. Louis College of Physicians and Surgeons in 1908. Dr. Walker later did postgraduate work at Tulane University, New Orleans; Crile Clinic, Cleveland; Mayo Clinic, Rochester, Minn.; and clinics in New York City. For forty-three years, beginning in 1908, Dr. Walker engaged in active practice in Wichita Falls. In 1950 he moved to Rocksprings, where he had a limited practice until shortly before his death. He specialized in surgery.

Dr. Walker was for many years city and county physician of Wichita Falls and Wichita County. He was chairman of the board of directors of Wichita General Hospital, Wichita Falls, from its opening until 1925, when he helped to found the Walker-Hargrave Hospital of that city, now Bethania Hospital. During World War I Dr. Walker served in the United States Army as a major with the British Expeditionary Force, and after the cessation of hostilities he received a commission from former President Herbert Hoover to survey the health conditions of children in Czechoslovakia and Poland.

Long active in medical organizations, Dr. Walker served the Wichita County Medical Society two terms as president and two terms as secretary; he was affiliated with the state and national organizations as well, being elected to honorary membership in the Texas Medical Association in 1951. He also belonged to the Royal Academy of Physicians and Surgeons. A member of the Baptist Church, Dr. Walker was a lifetime member of the Masonic Lodge and a member of the Shrine. He was fond of fine horses and dogs and enjoyed hunting and experimenting with the cultivation of beautiful flowers.

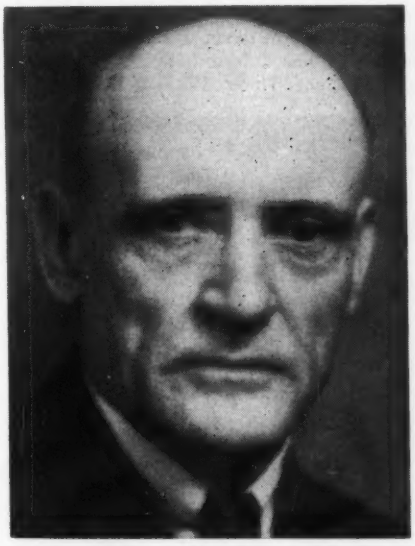
In Wichita Falls on January 12, 1910, Dr. Walker married Miss Vella V. Raney, who survives, as do his two sisters, Mrs. C. Lane, Clarendon, Texas, and Mrs. Winston Sherwood, Gainesville, Texas.

S. A. WOOLSEY

Dr. Samuel Albert Woolsey, Austin, Texas, died August 28, 1953, in that city of myocardial infarction.

Born August 20, 1882, in Round Rock, the son of Samuel M. and Eliza C. (Cloud) Woolsey, Dr. Woolsey attended Hutto, Georgetown, and Leander schools and the University of Texas Medical Branch before receiving his medical degree from the College of Physicians and Surgeons, Memphis, in 1909. He interned at Memphis City Hospital and then moved to Austin, where he practiced until his retirement in 1949. He was city health officer from 1913 to 1920 and then served as assistant for several years. He was a staff member of Breckenridge Hospital and a staff member emeritus of St. David's Hospital. After active service as a first lieutenant in the United States Army Medical Corps during World War I, Dr. Woolsey became a captain and later a major in the Medical Corps of the Texas National Guard.

A member of the American Medical Association and an honorary member of the Texas Medical Association through the Travis County Medical Society, Dr. Woolsey was a thirty-second degree Scottish Rite Mason, a Shriner, and a member of the Ben Hur Shrine Chanters and Texas Shrine Chanters Association as well as the American Legion and Phi Rho Sigma medical fraternity. He was an honorary steward in the First Methodist Church of Austin and a member of the church choir for many years.



DR. SAMUEL A. WOOLSEY

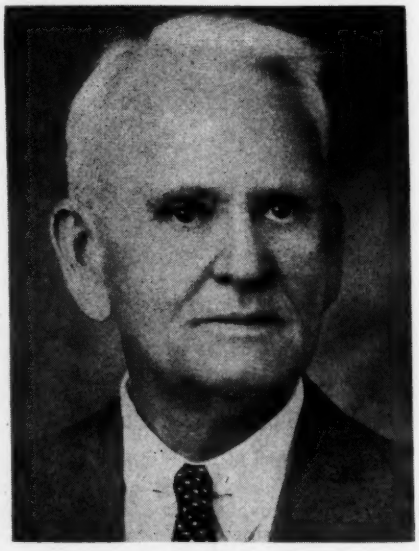
Dr. Woolsey in 1905 married Miss Loula E. Murray, who died in 1932. Their daughter, Mrs. Ben Lee Chote of Austin, survives. Also surviving Dr. Woolsey is the former Miss Katherine M. Barry, whom he married in August, 1937, and their son, Sam A. Woolsey, Jr., Austin; two sisters, Mrs. J. E. Howze, Austin, and Mrs. O. E. Faubion, Temple; and two brothers, E. R. Woolsey and Emmett R. Woolsey, both of Leander.

P. M. PAYNE

Dr. Peel Morphy Payne, Asherton, Texas, died in his home from nephritis after a lengthy illness, on August 19, 1953.

Dr. Payne was born July 4, 1870, in the Hunt County

community of Payne's Store, the son of Dr. John Haynes and Vernada Ann (Bonner) Payne. He received his academic training in a private school, Glendale Male Institute, Terrell, and in Terrell High School; he also attended Dallas Business College. Dr. Payne was graduated from Memphis Hospital Medical College in 1893, from which in 1948 he received a certificate of merit for fifty-five years of service to humanity. After serving as resident in surgery at the Eye, Ear, Nose, and Throat Hospital, New Orleans, he began his medical career in Terrell, Texas, where he remained for one year. "El Peregrino," as Dr. Payne called himself because of his wanderings in subsequent years, lived for long periods in Mexico and Louisiana. Also, for shorter times, he practiced in several Texas towns, including Austin, Redwater, Flusher, Beeville, Carrizo Springs, Comfort, and finally Asherton. In the last-named community he was active in medical practice from 1946 until he became ill in November, 1951. Although he engaged in general practice, Dr. Payne had interests in public health, industrial medicine, and diseases of the eye, ear, nose, and throat. He served as director of public health units in Louisiana for fifteen years and was city health officer of Asherton at the time of his death, although he had been unable to serve in recent months. Dr. Payne did postgraduate work in New Orleans Polyclinic in 1895 and in the Eye, Ear, Nose, and Throat Hospital of the same city in



DR. PEEL MORPHY PAYNE

1921. During World War I he worked with the YMCA, for eleven months during the influenza epidemic at Kelly Field, after he was refused for military service because of physical disability.

Elected to honorary membership in the Texas Medical Association in 1950, through LaSalle-Frio-Dimmit Counties Medical Society, Dr. Payne also was a member of the Southern and American Medical Associations. He was a life member of the American Public Health Association. A member of Phi Chi medical fraternity, Dr. Payne was active in Lions Club. He was a Mason and a member of the Episcopal Church. Dr. Payne, a charter member and former vice-president of the Scottish Society of New Orleans, was especially interested in the study of foreign languages (Latin, Spanish, and French) and in the life and poems of Robert Burns. An article by him, "The Democracy of Robert Burns," appeared in the Houston *Chronicle*, and he recorded his early experiences in and around New Orleans in a manuscript entitled "El Peregrino." Listed in "Who's Important in Medicine," Dr. Payne was on the program of the White House Conference on Child Health and Protection in 1931.

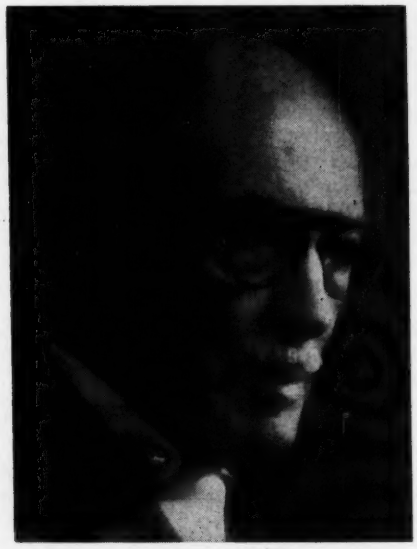
Dr. Payne married Miss Marie Daniels in Austin on August 18, 1898; surviving him are their children, Mrs. G. W. Shield, San Angelo, and Mrs. H. C. Londoner and Miss Frances Payne, both of Los Angeles. Dr. Payne also is survived by his wife, the former Mrs. W. J. Pilgree, whom he married June 11, 1947, in Uvalde; one sister, Mrs. Della Wimberley, Tempe, Ariz.; and three grandchildren.

M. B. STOKES

Dr. Merle Benefiel Stokes, Houston, Texas, died in a Houston hospital September 11, 1953.

Born September 10, 1880, in Lebanon, Ind., the son of Wilson R. and Harriett (Benefiel) Stokes, Dr. Stokes attended high school in Lebanon and Wabash College in Crawfordsville, Ind., before receiving his medical degree from Rush Medical College, Chicago, in 1909. After an externship at Presbyterian Hospital in Chicago, he interned at City Hospital in Akron, Ohio, and moved in 1910 to Houston, where he remained until his death. He served with the United States Army Medical Corps at the base hospital at Camp Travis, 1918-1919.

A member of the Texas Medical Association and the American Medical Association for many years through the



DR. M. B. STOKES

Harris County Medical Society, Dr. Stokes was certified by the American Board of Surgery and belonged to the American Association for Surgery of Trauma, the American College of Surgeons, and the Texas Surgical Society. He was secretary of the Harris County Medical Society in 1934-1935 and president in 1936; he was president of the Postgraduate Medical Assembly of South Texas in 1948. His college fraternity was Phi Gamma Delta and his medical fraternity, Nu Sigma Nu. He was a member of the Presbyterian Church.

Dr. Stokes married Miss Eva Blanch Van Nuys in Lebanon on April 16, 1910. She preceded him in death. His brother, George Stokes, South Berlin, Mass., and three nieces survive.